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THE LAURENCE - MOON - BIEDL SYNDROME
A PATHOLOGICAL REPORT

BY

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REFERENCES to the Laurence–Moon–Biedl syndrome have appeared in the literature of recent years with increasing frequency.

In 1931 I published a case showing the typical characters in a girl of five years of age. She was big, obese, dull mentally, had symmetrical polydactyly and syndactyly of the feet, and retinitis pigmentosa with a squint and nystagmus. There was a family history of polydactyly and syndactyly, and a brother, who died soon after birth, probably exhibited the same syndrome.

The child made satisfactory progress for two years, growing brighter and less torpid, and becoming much more interested in things. She grew about four inches, and her weight remained stationary while under treatment with whole gland pituitary tablets. Unfortunately she fell a victim to an epidemic of measles, developed intestinal and pulmonary symptoms, and died on the fourteenth day of her illness with a sudden rise of temperature to 106° F.

An autopsy was performed within twenty-four hours of death. The skull was symmetrical, broad at the parietal eminences and very thin, the thickness of the bone being from 1 to 2 mm. The greatest transverse diameter was 13.3 cm., and the anteroposterior diameter 18 cm. There were no bosses or indentations, and the base of the skull appeared normal. The brain had been fixed in situ by formalin introduced by cisternal puncture soon after death ; it weighed on removal 1,300 gm.

Of the other organs, the lungs were very congested; the heart appeared normal; the liver was congested and showed early nutmeg change; the spleen was congested but normal in appearance. The uterus, ovaries and tubes were unusually small, even for a child of seven; their microscopical structure was normal, but primordial follicles were very few. Both kidneys were markedly abnormal, with irregular deep lobulation and some capsular adhesions. They were congested. Sections showed a foetal appearance, with thick bands of fibrous tissue forming the lobulations. The thyroid was normal in size and appearance ; it had well-formed vesicles full of colloid. The thymus was normal in size, but microscopically it consisted of small islets of thymic tissue separated by thick, fibrous bands, with

much fat; Hassall's corpuscles were small and very few in number. The adrenals were normal in appearance; they gave no fuchsinophil reaction with the Ponceau-fuchsin stain. The pituitary body was very small; serial sections were made through it in the sagittal plane. The posterior lobe was small but normal in appearance; the anterior lobe showed great vascularity; the sinuses were unusually obvious and dilated, and seemed more numerous than normal. In every section there appeared to be a high proportion of basophil cells throughout the lobe, but there was no adenoma formation. There was a relative paucity of eosinophil cells. No colloid was seen. There was no extension of pars tuberalis up the stalk.

External examination of the brain showed marked and universal congestion of the membranes and vessels. Small thrombi appeared in some of the smaller vessels. At the base of the brain was evidence of some hæmorrhage, and on the vertex were two small subarachnoid hæmorrhages, each about the size of a penny. The convolutional pattern was normal, but the frontal poles were shrunken. Vertical section of the brain gave a normal picture, with no evidence of internal hydrocephalus.

For microscopical examination, serial sections were made of the base of the brain from the anterior part of the chiasma to the midbrain. Sections were stained by hæmatoxylin and eosin, and intermediate sections by Nissl, Loyez, Mallory's phosphotungstic acid hæmatoxylin, and by the Gross-Bielschowsky methods. Comparable series were made of several other brains.

Sections of the hypothalmic region showed the central grey matter with an abundance of small nerve cells, which stained rather faintly. Towards the pituitary stalk these cells were more numerous and closely packed, without showing any other characteristics.

The paraventricular nuclei, lying roughly parallel to the walls of the third ventricle, were well marked with their rather large, closely packed cells. The nuclei were not sharply defined, the cells tending to scatter at the periphery. In this plane the cells appeared mostly multipolar, with a few bipolar cells, having eccentric nuclei which is normal, and rather faintly staining cytoplasm, except that in two or three cells in each section it was very deeply stained.

The supraoptic nuclei were well defined as two groups, lateral and mesial, joined by a narrow band of cells. In size, shape, and staining qualities the cells resembled those of the paraventricular nuclei. A few cells showed the same deep staining, which was not seen in any of the other brains examined, and may be due to the fixing method.

The tuberal nuclei in this plane appeared in two groups, a lateral reniform and a mesial smaller round nucleus, rather difficult to demonstrate, but showing clearly with some staining methods. They formed paler, emptier areas, relatively avascular, with few cells. These were small, with well-stained nuclei, the cytoplasm forming a faint halo, sometimes being filled with a pale yellowish substance. In the periphery of the nuclei were a few larger pale cells, easily distinguished from the well-stained cells of the adjoining premamillary group. In this brain the tuberal nuclei seemed emptier than normal, with fewer of the large cells, but there was no evidence of cell degeneration or destruction.

Scattered in the central grey matter between the paraventricular and supraoptic nuclei were some clusters of 6–8 large, well-stained cells, usually closely related to a blood vessel, but apparently not connected with the main cell groups, or with each other. They were present in all cases examined, the cells resembling those of the well-marked premamillary group.

The mamillary body was well marked and showed no abnormality. Similarly, other cell groups, the well-marked basal nucleus of Meynert and the nucleus substantia innominata, which are considered by many as identical, the corpus Luysii, substantia Nigra, and the midbrain, appeared quite normal.

In sections stained by the Loyez method, the myelinated nerve fibres appeared

to be normal in distribution and quantity. The optic tracts appeared to be well myelinated and normal in size. In the central grey region there were a few rare myelinated fibres, apart from the commissure of Meynert, and constantly two small fibre groups were seen in the lateral part of the supraoptic nucleus.

This stain showed the distribution of capillaries very clearly in this congested brain. The paraventricular and supraoptic nuclei showed a thick network of capillaries among the cells, and the massa intermedia (nucleus reuniens) was considerably congested. The tuberal nuclei were almost free of capillaries, which were scattered throughout the central grey matter. The pituitary stalk showed much capillary congestion. In the other parts of the region the congestion was not so marked.

In sections stained by silver the unmyelinated fibres of the region were shown. Fine fibres were found running erratically throughout the hypothalamus, but there were certain well-marked tracts. A tract appearing to arise in the paraventricular nucleus curved towards the lateral part of the infundibulum ; here it intermingled with fibres appearing to come from the lateral part of the supraoptic nucleus. From this mingling some fibres appeared to pass down into the pituitary stalk, and others to cross to the opposite side. The direction and connections of these fibres with cells could not be confirmed with any certainty. Fibres appeared to link up the paraventricular and lateral supraoptic nuclei, and some fibres lying parallel to the ventricular wall could be traced to the vicinity of the anterior commissure. The tuberal nuclei were outlined by fibres most marked on the lateral aspect, but their connections could not be followed in this plane. No fibres could be seen in association with the mesial part of the supraoptic nucleus, or the odd cell groups. The staining of these fibres is difficult as they are very slow to take up the silver, but the quantity and distribution of fibres in this case was comparable to that of the control brains.

The neuroglia showed no abnormal development. The subependymal glia of the third ventricle was somewhat thickened near the lower pole of the paraventricular nucleus, corresponding to an area where the ependyma itself is deeper than elsewhere. There was also a framework of neuroglia fibres in the more superficial parts of the supraoptic nuclei ; these findings were constant in all cases. The tuberal nuclei were free of neuroglia, and in the Mallory stain appeared to have a light pink, rather foamy ground substance, making the nuclei particularly easy to define.

Sections were also taken from the cortex (frontal, Rolandic and occipital), the basal ganglia, pons and medulla. The outer parts of the cortex in all areas was congested, with small extravasations. The cells were for the most part normal in size and number, and the arrangement showed no gross distortion, though there were small irregularities in places. In the frontal region the shrinkage seemed to affect the white matter rather than the grey, but no disturbance of myelination was seen. All other regions were normal in appearance, except for the excessive presence of globules of free fat throughout the globus pallidus.

The pineal body appeared normal in size. The eyes, unfortunately, were not preserved.

In the case here described it is apparent that there are widespread abnormalities ; developmental defects of the skeleton, kidneys and probably eyes, and abnormalities in the pituitary body, thymus and gonads. The findings in the brain are slight, a diminution of volume of the gyri of the frontal lobe, and a paucity of cells in the tuberal nuclei. Our knowledge of the histopathology of the hypothalamic nuclei is still scanty, and the peculiarities of shape and staining reactions demand caution in interpretation ; while as the connections of the nuclear masses are by unmyelinated fibres, it is difficult to demonstrate with certainty the integrity of the fibre-tracts.

The syndrome called generally by the names of Laurence and Biedl has been

described fully by several authors, who have given various theories of its causation in the absence of pathological investigation. In this country Cockayne, Krestin and Soresby (1935) have discussed the genetic aspect, and compared the condition with juvenile amaurotic idiocy ; the pathological findings do not suggest any relationship. The theory suggested by Raab (1924) and others that some foetal occurrence causes an internal hydrocephalus affecting the hypothalamic nuclei is manifestly incorrect. Van Bogaert and Borremans (1936) in a recent paper give the autopsy findings in a case they include in this group. Their case had the characteristics of family incidence, mental defect, obesity, genital dystrophy and retinitis pigmentosa, but the skeletal defect was a hyperostosis of the inner table of the frontal bone, a condition which has been previously associated with obesity without the other symptoms. They found the brain normal, but the pituitary body showed stasis of the vessels with no change of cell structure. They found a necrotic area in the pituitary stalk. They do not mention any other organs.

The multiplicity of anomalies in the absence of gross cerebral deformity seems to confirm the view of L'Hermitte and Bollack (1936), that it is a germinal affection " totus substantiæ." The distinction between cerebral and pituitary adiposity seems one rather of function than of anatomical change, so slight a change in the tuberal nuclei can scarcely be credited with so gross a metabolic disorder. There seems, however, to be some analogy with the syndrome of pituitary basophilism in the distribution of the fat, the colour of the cheeks, a tendency to raised blood pressure, perhaps albuminaria, and the defect of genital function. This is possibly emphasised by the large proportion of basophil cells in the anterior lobe of the pituitary body. The genetic aspect of the syndrome has obvious importance in any discussion of its causation. The stasis found throughout was probably the result of the terminal condition and not associated with the syndrome under discussion.

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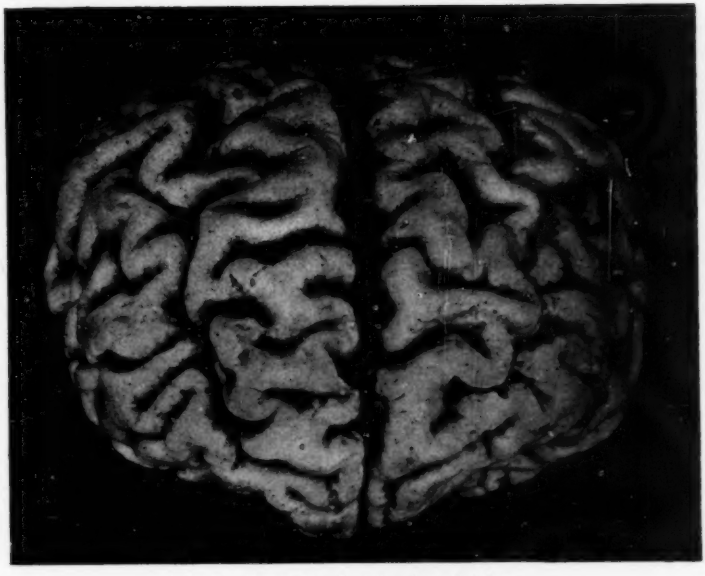

Fig. 1.—Frontal poles showing shrinkage.

Fig. 2.—Part of the wall of the third ventricle, Mallory stain, showing subependymal gliosis near the paraventricular nucleus.

Fig. 3.—Supraoptic nucleus showing capillary distribution, Loyez stain.

Fig. 4.—Hypothalamic region, stained by Gross-Bielschowsky method, showing the paraventricular and supraoptic nuclei and the unmyelinated fibre tracts A.—Paraventricular nucleus. B.—Supraoptic nucleus. C.—Infundibulum.

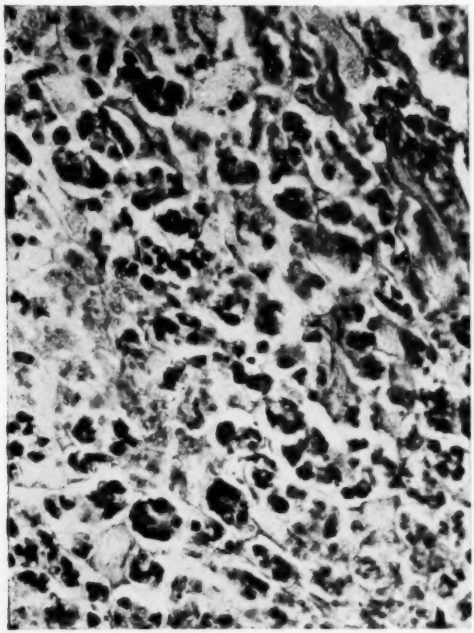

Fig. 5.—Anterior pituitary, showing basophilia, stained by methylene blue and eosin.

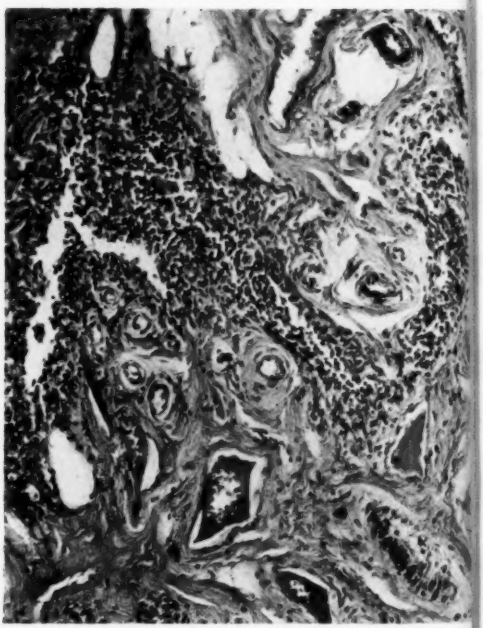

Fig. 6.—Thymus, showing fibrosis.

AN EXAMPLE OF STATUS MARMORATUS OF THE CEREBRAL CORTEX

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THE phenomenon of hypermyelinization has received much attention from neuropathologists since C. Vogt's (1911) original description of status marmoratus of the corpus striatum. It is now well established that hypermyelinized fibres in the form of plaques or as the peculiar networks which first suggested the name " marbled " may be present in many parts of the nervous system, though they are most common in the basal ganglia and cerebral cortex. A review of the recorded cases shows that several ætiological factors may be operative in the production of this remarkable state (Hallervorden, 1930). Thus, hypermyelinized plaques have been found associated with traumatic lesions (Freund and Vogt, 1911 ; Pfeiffer, 1925 ; Scholz, 1924), general paralysis (Bielschowsky, 1924), disseminate sclerosis (Wertham, 1924), von Recklinghausen's disease (Bielschowsky, 1924), senile dementia (Stief, 1924), telangiectiasis of the cortex (Bielschowsky, 1924), carbon monoxide poisoning (Spatz, 1931), febrile illness in infancy (Anton, 1896 ; Scholz, 1924 ; Case, 1934 ; Lowenberg and Malamud, 1933), and birth injury (Meyer and Cook, 1936). Familial instances of status marmoratus of the corpus striatum are also on record (Anton, 1896 ; Scholz, 1924 ; Vogt, 1920). The pathogenesis of the condition is obscure. Since hypermyelinization is almost invariably accompanied by some degree of gliosis, there are grounds for believing that a perversion of glial function has led to the abnormal myelin production. Considerable uncertainty, however, still exists as to the mode of origin of the abundant axis cylinders found in the plaques. Shrinkage of the tissues might conceivably produce the linear hypermyelinized scars so frequently seen in the cerebral cortex, but it is difficult to imagine how such a mechanism could produce the distinctive networks of a true status marmoratus. Bielschowsky's (1924) suggestion that previously naked axis cylinders have become myelinized also fails to explain the excessive number and peculiar arrangement of the fibres. Most authors seem to have followed Scholz (1924) in interpreting the abnormal axis cylinder development as a regenerative phenomenon, despite the fact that in experimental animals only ephemeral and abortive regenerative activity has been observed. The experiments of Borst (1904) have been cited as proving that a new growth of myelinized fibres can actually occur after cerebral lesions, but, as Cajal (1928) has pointed out, his findings are open to other interpretations and he did not employ neurofibrillar staining methods.

I have thought the following case worthy of record since, among other points of interest, it exemplifies the association of status marmoratus with certain cortical abnormalities of prenatal origin.

Personal Case

The patient, a male aged at death 27 years and 2 months, died at Stoke Park Colony on 18th April, 1935, from Banti's disease after an illness of approximately

sixteen months' duration. He was an illegitimate child and nothing is known of his life prior to admission to the Colony. Examined in 1928, he was classified as an idiot having a mental age less than 3 years. The records state that he was unfit for any but the simplest occupation, was dirty in habits, and was very excitable and inclined to be dangerous to others. He suffered from grand mal, was " partially blind," his speech was limited and indistinct and some weakness was observed in the legs. The Wasserman reaction in the blood was negative (November, 1934).

A neurological examination made shortly before death gave the following information. The pupils reacted to light and on convergence. The tendon reflexes in the upper limbs were very brisk and equal. The upper abdominal reflexes were not obtained. The lower abdominal reflexes were present and equal. The cremasteric reflexes were absent. In the lower limbs, the knee-jerks were equal and exaggerated, Strümpel's sign being positive. The ankle-jerks were equal and increased. There was no knee or ankle clonus. The plantar reflexes were flexor. There was no record at any time of athetoid movements.

Pathological Examination

At the autopsy which was made 16 hours after death the diagnosis of Banti's disease was confirmed. At the time of removal of the brain no abnormal adhesions between the dura mater and the calvaria were found and there was no sign of old sinus thrombosis. The meninges appeared to be normal.

The brain was markedly micrencephalic. Measurements obtained from the right hemisphere gave the following readings : length, 140 mm. ; breadth, 54 mm. ; height, 100 mm. A comparison between this brain and the normal brains in the collection at Stoke Park Colony showed that its size corresponded approximately to that of an infant of a year old. This reduction in total volume was in large measure due to the especially small size of the parietal and occipital lobes. After stripping the leptomeninges, it was seen that both hemispheres exhibited gross structural abnormalities. On both sides the Rolandic sulcus was laid down in two parts, and in the left hemisphere the upper part of the postcentral gyrus seemed to be represented by a thin, ribbon-like structure which was concealed in the depth of the central sulcus. This peculiar formation took its origin from the posterior wall of the precentral gyrus at a point above the general level of the floor of the Rolandic sulcus. The presence of numerous small gyri interspersed between gyri of more normal size gave the upper part of the parietal lobe a markedly abnormal appearance. The mesial surface and the upper part of the outer surface of the occipital lobes were similarly affected. The calcarine fissures could be identified, but the arrangement of the neighbouring gyri was confused by an over-production of small, shrunken, and apparently sclerotic convolutions. These irregularities were only approximately symmetrical in the two hemispheres. A more detailed examination of these changes gave the impression that while some of the appearances presented by the larger gyri might be attributed to the distorting influence of sclerosis (ulegyria), this factor could not account for all the abnormalities found in the convolutional pattern. A malformation of the cortex in the form of a partial microgyria seemed to have formed the basis of this complex arrangement.

On sectioning the brain it was seen that the white matter of the centrum ovale was very thin subjacent to the abnormal portions of the cortex, the lateral ventricle in this position being dilated. Many of the gyri presented a constriction in their basal portions. The brain tissue in such situations was extremely tough and evidently sclerosed. In the less sclerotic gyri, partial shrinkage had produced an effect somewhat resembling candle gutterings.

In the dorsal part of the putamen an abnormal network of white fibres characteristic of status marmoratus could be distinguished macroscopically. The corpus callosum

was noticeably small and at a point just anterior to the splenium narrowed to a depth of about 2 mm.

Cerebral Cortex.—The abnormal construction of the small and distorted gyri in the parieto-occipital part of the cortex may most conveniently be explained by reference to the illustrations. Fig. 1 shows typical gyri with a variety of myelin abnormalities, the following points being especially noteworthy :

(*a*) The occurrence of several small gyri developed upon one parent stem (see also Fig. 3).

(*b*) Typical retiform status marmoratus in the larger gyri.

(*c*) Diffuse hypermyelinization in three small convolutions *a*, *b*, *c* (Fig. 8). The myelinated fibres in these areas (as also in Fig. 6) were present in dense whorls and practically confined to the grey matter, the central core of the gyrus containing very few axis cylinders. The latter fact was confirmed in Bielschowsky preparations. The molecular layer in such gyri showed an abnormal wealth of myelinated fibres.

Sections stained for glial fibres by Holzer's method showed that the parts of the cortex where myelin was scanty or absent, more particularly the constricted basal parts of the gyri, were the seat of an extremely dense sclerosis. Small areas of rarefaction and porus formation were frequently seen (Fig. 2). The blood vessels in such areas showed intense fibrotic thickening (Fig. 11). The myelinated parts of the central cores showed a fairly dense isomorphous gliosis. In the areas of the grey matter exhibiting hypermyelinization a much finer gliosis was found, which was usually only demonstrated with difficulty (Fig. 14) and which was often invisible under low-power magnification. In some areas of status marmoratus repeated staining of sections failed to demonstrate any glial fibres in the grey matter, although a dense marginal gliosis was usually observed together with obvious proliferation of glial fibres in the central core of white matter.

An interesting glial abnormality was found in sections taken from the macroscopically normal temporal pole. Special staining showed the presence of numerous very large fibre-forming astrocytes in the grey matter of the cortex, chiefly in the lower layers (Fig. 13). Other abnormal glia cells were characterized by their coarse, fleshy processes and large, deeply-staining cell bodies. Figs. 3 and 4 illustrate many of the peculiarities described above, and in addition, a remarkable abnormality in the arrangement of the myelinated axons composing the central parent stem of a cluster of small gyri. The fibres derived from these gyri are present as two strands on either side of a central area devoid of myelin. The glial picture is practically the reverse of the myelin preparation.

Sections of the cortex stained for nerve cells showed a variety of abnormalities. The most common finding in the area showing status marmoratus was a patchy distribution of the cytons. In some of the smaller gyri, for example, those in which diffuse hypermyelinization was present, the nerve cells were arranged in clusters and columns with no attempt at differentiated lamination (Fig. 7). The majority of these cells were well-formed pyramidal cytons, some being of disproportionately large size compared with their fellows. Bielschowsky's staining revealed a very rich axis cylinder development confined to the grey matter. The impression given by such gyri was not that of a normal cortex which had undergone pathological change, but rather that of a malformation. In contrast to this latter type of gyrus, those parts of the cortex in which a severe degree of sclerosis was present showed a nerve-cell picture typical of ulegyria (the " granular atrophy " of Spatz). Such changes were most frequently encountered in the walls and basal portions of the gyri, the line of demarcation between the atrophic part of the gyrus and the bulbous neurone-bearing crown being often abrupt. In the upper part of the Rolandic motor area there was a typical status marmoratus (Fig. 5). Sections taken from this area and stained for nerve cells showed that the deepest layer of the cortex (L.VI) was in parts entirely absent, its place being taken by proliferated neuroglia. The Betz cells of L.V thus

represented the lowest recognizable limit of the grey matter (Fig. 9). These cells were numerous and most were of fully adult size. Some displayed a marked degree of apolarity and many had assumed a circular or ovoid shape, the nucleus being displaced in a lateral direction. Sections stained by Bielschowsky's method showed that the parts of the cortex exhibiting status marmoratus also showed a dense network of fine axis cylinders. One could distinguish several examples of Betz cells possessing numerous short processes. In favourable sections these latter could be shown to break up into finer branches contributing to the surrounding rich fibrillary network of the status marmoratus (Fig. 12). In the small ribbon-like upper part of the postcentral gyrus the nerve cells were confined to the small bulbous crown of the convolution, the remainder of the gyrus being composed of dense glial fibres and containing few myelinated fibres. A much finer gliosis was present in the neurone-bearing area where a diffuse hypermyelinization of fibres was observed (Fig. 14).

Two other abnormalities were encountered which are worthy of mention. One of these is shown in Fig. 10, a low-power view of the visuosensory cortex. The lower layers of the cortex were spread out into two thin bands that contrasted markedly with the denser aggregation of nerve cells in the outer part of the gyrus, from which they were separated by an appreciable depth of tissue containing only glial nuclei. The other abnormality was met with in one block of tissue removed from the macroscopically abnormal parieto-occipital region. This was the presence of a heterotopic collection of some 20 abnormally shaped nerve cells lying in the white matter at a point approximately half-way between the ependyma and the small sclerotic cortical gyri. This heterotopia was sufficiently large to show as a pale area in myelin preparations.

An examination of cortical areas remote from those displaying the gross abnormalities recorded above showed little to distinguish them from those commonly encountered in cases of low-grade mental deficiency. From the point of view of size, the nerve cells of the supragranular layers of the temporal and frontal lobes were moderately well developed, but their arrangement was in general typically irregular. No defect was found in the pyramidal cell layer of the cornu ammonis.

The microglia in the cortex showed no abnormality.

The oligodendrocytes in the areas of status marmoratus showed acute swelling.

Cerebellum.—The only abnormality found in sections taken from the vermis consisted in the occasional presence of " torpedoes " in the axons of the Purkinje cells. There was a slight gliosis of the white matter of the folia.

Putamen.—The presence of a well-marked status marmoratus which had been observed macroscopically in the dorsal part of the putamen was confirmed when sections were stained for myelin (Fig. 15). Bielschowsky's method showed in the areas of hypermyelinization a corresponding number of fine axis cylinders. As in the cortex, no bulbs, spirals, or other phenomena suggestive of regeneration were seen. There was a fine gliosis in these areas, which was demonstrated only after repeated efforts with the Holzer stain. Corresponding pictures of myelin and glia were not obtained. A diminution in the numbers of ganglion cells was observed and an increase in small glial nuclei, many of which proved to be those of swollen oligodendrocytes. The microglia showed no abnormality, nor were the blood vessels increased in number. The external capsule showed an intense gliosis and was densely myelinated. The claustrum and some parts of the insular cortex also showed areas of hypermyelinization.

Thalamus.—No status marmoratus was found in the thalamus. The subependymal region showed an increase of fibrous neuroglia. The glial fibrosis seemed to be densest in the neighbourhood of the terminal vein.

Head of the Caudate Nucleus, Globus Pallidus, Midbrain, Pons, Medulla, and Spinal Cord.—No abnormality in myelin or nerve cells was found in these parts of the nervous system.

External Geniculate Body.—It was about one-third the size of a normal control

sectioned in a similar manner for purpose of comparison. Its cell content was also notably inferior to the normal. The characteristic structure of the nucleus could, however, be distinguished.

Discussion

The foregoing microscopical examination has shown that this case is one of considerable complexity, and it is by no means easy to trace the temporal sequence of the various abnormalities described. There are several indications, however, that some of the cortical changes had resulted, in the first instance, from a disturbance of development occurring during fœtal life. In contrast to the usual case of ulegyria in which previously normal convolutions have become shrunken and distorted by sclerosis, the fissuring of this brain was abnormally complex owing to the presence of a multiplicity of small gyri. The microscopical examination of such areas afforded many instances of gyri the abnormal cytoarchitectonics of which could not be ascribed simply to destructive processes. It would therefore seem justifiable to regard such gyri as primarily microgyric. The presence of a heterotopic collection of nerve cells in the subcortical white matter strengthens this supposition. This picture of malformation, however, was complicated by widespread evidence of degenerative process, involving not only microgyric areas but larger and more adult convolutions also. These atrophic changes were most marked in the walls and basal portions of the gyri, leaving the bulbous, neurone-bearing crowns relatively intact. This regional distribution is not uncommon in examples of atrophic lobar sclerosis without status marmoratus, and is probably due to anatomical factors of vascular supply. The findings in the present case thus seem to admit of two different interpretations : the first, that a pathological process leading to sclerosis has been engrafted upon a pre-existing malformation ; the second, that both the appearances suggestive of malformation and the atrophic changes have arisen as the result of a pathological process acting upon the cerebral cortex during an early stage of its development. The latter explanation has the merit of accounting for the observed facts without involving a double pathology.

The main interest of this case lies in the widespread hypermyelinization present in the cerebral cortex. As has been pointed out in the introduction, the most difficult problem raised by a study of status marmoratus is not the mechanism by which the axis cylinders have become myelinated but the origin of the axis cylinders themselves. As in the striatum, so in the cerebral cortex one is confronted with the paradoxical finding of an excessive production of myelinated fibres together with a diminution of the total nerve-cell content of the area involved in the change. In the present case the axis cylinders in the areas of status marmoratus showed none of the bulbs, spirals, or spools that some investigators consider to be the result of regenerative processes. There is another possible explanation as to the origin of these abnormal strands and networks of axis cylinders which seems to have escaped the notice of previous observers. The fibres may have arisen not as the result of true regeneration but by a process analogous to that described by Cajal (1928) as compensatory collateral hypertrophy. In Cajal's experiments section of the axon of pyramidal cell distal to the origin of the initial collaterals resulted in the compensatory hypertrophy of these collateral branches. This reaction, which was named " arciform transformation " of the nerve cell, led to the development of a neurone which was " a perfect reproduction of a cell with a short axon except that the terminal arborization is composed of hypertrophic branches which involve an enormous part of the cortex." It is not without significance that those gyri showing the densest marbled state were those whose basal portions were affected by the most severe sclerosis and contained only a small number of axis cylinders. The hypermyelinization of the axis cylinders in the areas of status marmoratus may be attributed, as in other cases, to a perversion of glial activity. It was usually possible to demonstrate a slight excess of glial fibres

in these areas of the grey matter, a " partial sclerosis " in the sense of Scholz being sometimes present. The failure to find gliosis in some of the marbled areas may be due to technical faults, though it may be pointed out that Case (1934) and in one instance C. Vogt (1925) failed to find a glial proliferation corresponding to the myelin networks. Possibly the fact mentioned by Meyer (1936), that the degree of marbling tends to vary from section to section, may explain this discrepancy. Nevertheless, in the present case it would be inadvisable on the histological findings to speak of " myelinated scars " being present in the grey matter, though the white matter showed numerous examples of such formations.

From the clinical standpoint this case of status marmoratus is remarkable in that the patient did not exhibit during life the athetoid movements characteristic of this form of striatal abnormality. The explanation is probably to be found in the coincident involvement of the cerebral cortex, more particularly of the lower layers of the precentral gyrus, the integrity of which is a necessary factor in the exteriorization of complex hyperkinetic movements.

Summary

The pathological examination of the brain of an adult epileptic idiot showed extensive status marmoratus of the cerebral cortex and of the dorsal portion of the putamen.

The parietal and occipital lobes were fissured in an abnormally complex manner, and showed what was considered to be a combination of malformation and atrophic changes.

A short account of some of the problems of the pathogenesis of status marmoratus has been given. The suggestion is made that the wealth of axis cylinders present in some of the marbled areas in the present case owed their origin not to a true regenerative activity but to a process akin to the compensatory collateral hypertrophy of Cajal.

It is a pleasure to record my thanks to Professor Berry for permission to publish these notes and also to Dr. A. Meyer for his helpful interest in the pathological findings of this case. This work was carried out on behalf of the Burden Mental Research Trust.

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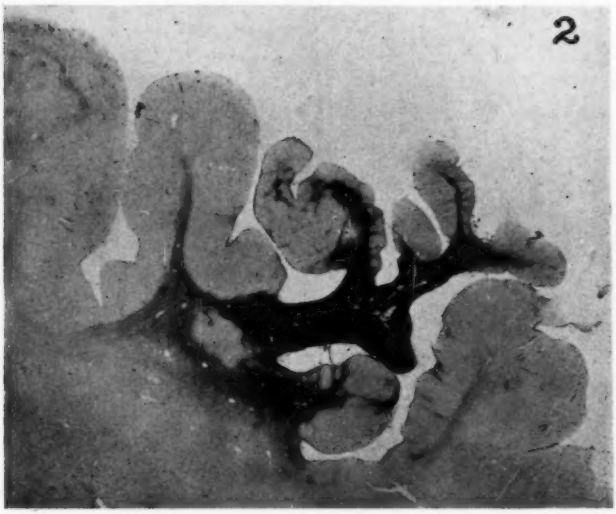
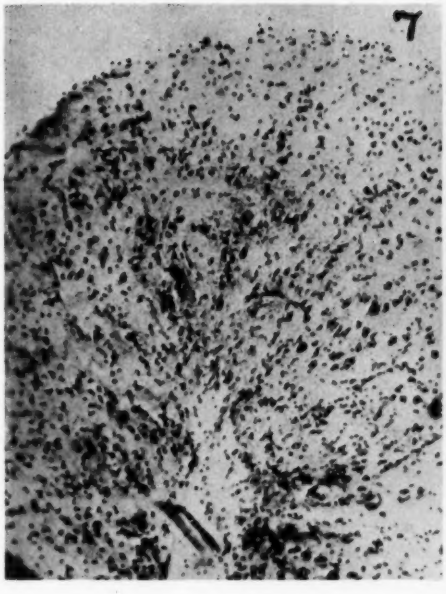


Fig. 1.—Gyri from the occipital region. The small gyri *a*, *b*, *c* show dense whorls of hypermyelinated fibres practically confined to the grey matter and molecular layer of the cortex. Anderson's modification of the Kulschitsky-Pal method; $\times 5\frac{1}{4}$.

Fig. 2.—Section taken from the same block of tissue as shown in Fig. 1. Holzer's stain; $\times 5\frac{1}{4}$.

Fig. 3.—Gyri from the occipital lobe. Kulschitsky-Pal stain ; ×4.
Fig. 4.—Section taken from the same block of tissue as shown in Fig. 3. Holzer's stain ; ×4.
Fig. 5.—Upper precentral gyrus (FA) showing status marmoratus in the cortex adjacent to the small atrophic postcentral gyrus. Kulschitsky-Pal stain ; ×3.
Fig. 6.—Diffuse hypermyelinization of the cortex ; the fibres are largely confined to the grey matter. Kulschitsky-Pal stain ; ×13.



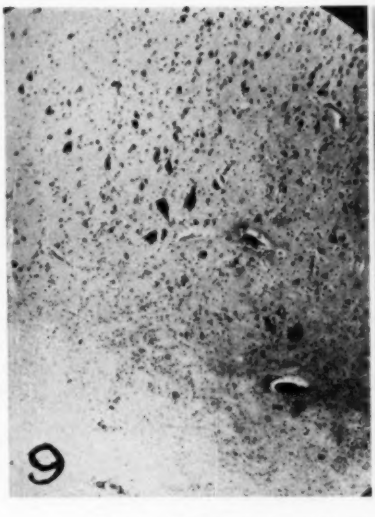
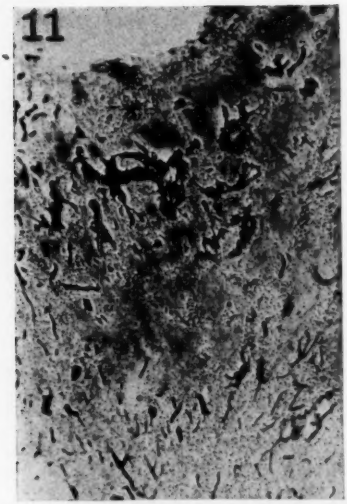
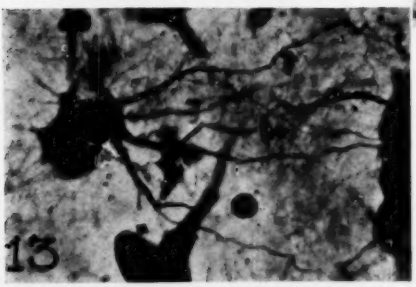


Fig. 7.—Nerve-cell picture of gyrus *b* in Fig. 1. Cresyl violet ; ×60.
Fig. 8.—As in Fig. 7. Kulschitsky-Pal stain, ×60.
Fig. 9.—Nerve-cell picture of motor cortex showing a polarity of Betz cells and absence of the sixth layer.
Fig. 10.—Low-power view of calcarine fissure showing abnormal arrangement of the deeper layers of the visuosensory area, typical basal constriction of the gyrus, and thinness of the subcortical white matter. *v*=ventricle. Cresyl violet; ×4.





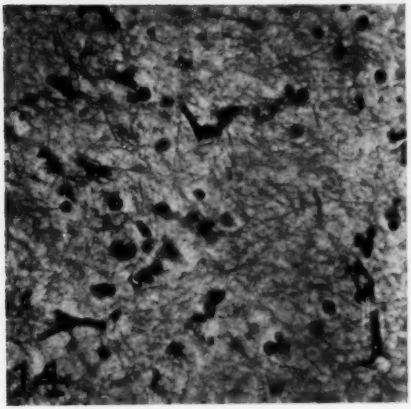


Fig. 11.—Fibrotic vessels in an area of severe sclerosis. Perdrau's stain; ×36.
Fig. 12.—Betz cell with branching process contributing to the axis cylinder network of an area of status marmoratus. Bielschowsky's stain; ×600.
Fig. 13.—Typical hypertrophic fibre-forming astrocytes found in sections from the temporal pole. Hortega's method for astrocytes; ×600.
Fig. 14.—Type of fine gliosis found in areas showing hypermyelinisation. Holzer's stain; ×270.
Fig. 15.—Status marmoratus of the putamen. Kulschitsky-Pal stain; ×4. (The section has been rather over-differentiated in order to show up clearly the marbled appearance of the putamen. Consequently, the cortex of the insula seems devoid of fibres.) The claustrum also shows hypermyelinization.

EPENDYMAL STREAKS AND ACCESSORY CAVITIES IN THE HUMAN OCCIPITAL LOBE

BY

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IT has long been known that the occipital horn is the most variable portion of the lateral ventricles of the human brain. Many authors have alluded to this curious fact. Thus, according to Testut and Latarjet (1929), there are numerous variations between individuals and, in one individual, between the right and the left sides. The different forms of the occipital horn, as observed by Davidoff and Dyke (1935), are as follows : (1) short and stubby ; (2) a straight, fingerlike projection of uniform calibre up to a rounded tip ; (3) tapering almost to a point ; (4) a bulbous tip ; (5) a tip shaped like a fish-tail. Finally, the occipital horn may be completely absent.

In the course of routine examinations of formalin-fixed brains from autopsies at the City Hospital, my attention was attracted to the presence of small, distinct cavities in the occipital lobe, caudal to the tip of the occipital horn of the lateral ventricle.

A search through the available literature revealed only four mentions of such accessory cavities in the occipital lobe. Retzius (1896) merely stated : " Schliesslich kommen auch Fälle vor, wo das hintere Ende des Hinterhorns von der Hauptabteilung abgeschnürt ist und eine besondere, kleine Höhle darstellt." Curran (1909) classified the occipital horns of 64 adult brains into three groups. In his group A the horn was patent to a point beyond the calcar avis (22 cases). In group B there was a complete constriction separating the occipital horn from its " isolated open tip " (24 cases). In group C the terminal portion was completely closed, resulting in a short occipital horn (18 cases). The ependyma was stated to persist in the occluded portions. Curran believed that the closure, whether partial or complete, results from the pressure of the rapidly growing calcar in late fœtal life. He suggested that ophthalmic migraine, epilepsy, and other transient cerebral disturbances might be due to temporary pressure on a partially closed occipital horn. In the case of a three-week-old infant who died in convulsions, the occipital horns were constricted and the tips were filled with a " cheesy " concretion. In the other cases, nothing was known about the age or history of the subjects. De Angelis (1929) reported the case of a large cavity in the right occipital lobe of a 31-year-old man, who died with a syndrome of intracranial hypertension. The cavity contained clear fluid and compressed the occipital horn, but did not communicate with it. Torkildsen (1934), among 11 cases in which the lateral ventricles were filled with paraffin and subsequently dissected, found one in which the occipital horn " was completely constricted with a cavity posterior to the constriction." No record could be found of microscopic study of these cavities or the surrounding tissues by any of the above-mentioned authors. It would seem difficult to affirm on the basis of gross observation alone that a cavity in the brain is a pinched-off portion of a ventricle and not a cavity secondary to some destructive lesion, such as ischæmic malacia, or to post-mortem change. The latter possibility must be seriously considered in connection with the specimens of Retzius, which were

fixed in bichromate or in weak formalin, and those of Curran, which came from the dissecting room. Furthermore, Essick (1915) discovered transient juxtaventricular cavities not lined with ependyma in human embryos, and Hochstetter (1919) suggested that in some cases cysts of the adult brain might be persistent embryonal cavities of this sort. This possibility would manifestly have to be considered before affirming that the occipital cavities were portions of the ventricle.

Since the first brain containing accessory occipital cavities was observed at the City Hospital laboratory of pathology, the condition was searched for in all specimens. This was done by making gross coronal or sagittal sections as close together as possible through the formalin-fixed brain. The absence of communication between the separate cavities, when present, and the occipital horn was established in each case by careful inspection, by transillumination, by attempts to pass a filiform bougie, by pouring water into the occipital horn and observing whether or not it passed into the accessory cavity, and finally by attempting to blow air from one cavity to the other while the specimen was submerged in water. It was deemed necessary to carry out these tests in every case, as the occipital horn sometimes narrows down to a minute slit, hardly visible, only to open out again caudally to form a terminal bulb. Without careful examination such forms might be taken for separate cavities. In every case where simple inspection suggested a distinct cavity, this was confirmed by these various tests. In one case with abundant intraventricular hæmorrhage the entire ventricular system was filled with blood, but the accessory cavity contained only clear fluid. In another case, an opaque mass, injected intracisternally, had filled all the ventricles but not the accessory cavity.

Of 50 unselected brains examined with this in view, accessory cavities were present in 16 cases. In five cases the cavities were bilateral. In 11 cases they were unilateral, 10 times on the right side and only once on the left side. The linear measurements of the gross cavities were as follows :

MEASURE	AVERAGE	EXTREMES	AVERAGE DEVIATION
Rostrocaudal extent.	5·4 mm.	8 to 1 mm.	2·4—
Transverse extent.	3·5 mm.	7 to 1 mm.	1·5
Dorsoventral extent.	2·9 mm.	6 to ½ mm.	1·4—
Distance from tip of occipital horn	10·2 mm.	15 to 5 mm.	2·1+

The relatively small number of cases precludes a more delicate statistical analysis. The occipital horn was usually short, broad, and relatively blunt-tipped when it was associated with an accessory cavity, longer and narrower in the absence of a cavity. The occipital horns were classified, wherever possible, into long and short types. Of the 50 cases in the series, three (one with and two without gross accessory cavities) had cerebral tumors which deformed the occipital horns beyond the possibility of recognizing their previous shape. In seven further cases, all without cavities, coming from young infants, the occipital horns were of an undifferentiated type which could not be classified as either long or short. This leaves 40 classifiable cases, with 80 occipital horns—15 cases with cavities and 25 without them. The distribution was as follows :

	SHORT GROUP	LONG GROUP
15 CASES WITH GROSS CAVITIES		
30 occipital horns :		
19 occipital horns with cavities.	17	2
11 occipital horns without cavities.	3	8
25 CASES WITHOUT GROSS CAVITIES		
50 occipital horns.	21	29

The total length of occipital horn, cavity, and intervening tissue was always about equal to that of the contralateral occipital horn in the unilateral cases.

The age-frequency distribution of the 16 cases with accessory occipital cavities

(6 months to 78 years) and that of the 34 cases without gross cavities (premature stillbirth of 8 months to 84 years) were similar in all but their lowest ranges. Cavities occurred in only one individual less than 16 years of age. This was an infant of 6 months. Of the 34 cases without cavities, one was 14 years old, one was 19 months, one 2 months, three were full-term babies, and two were premature babies, none of whom had accessory cavities. The youngest individual with accessory cavities was 6 months old. The distribution of other factors—sex, race, lesions of the central nervous system, lesions in other organs, cause of death—showed no significant difference between the cases with and those without gross accessory cavities. Of the 50 cases in the series, three presented congenital cerebral anomalies—dilated cavum septi lucidi, ventricular heterotopia of grey matter, congenital tumour of the thalamus, the latter in a new-born baby with arenia, absence of genitalia, and other anomalies. A fourth individual, with congenital atresia of the bile ducts, was a baby of 2 months. None of these four individuals with developmental anomalies had accessory cavities.

The cavities were constantly situated at a short distance lateral and ventral to the calcarine fissure, to which they bore the same relation as the terminal portion of a long and narrow type of occipital horn. In the larger accessory cavities the calcarine fissure corresponded to a fullness in the dorsomedial wall, representing the calcar avis. The collateral eminence was sometimes indicated in the floor of the cavity. The medial wall was thus always more or less convex in the larger cavities. The inferior wall was flat or slightly excavated. The posterior extremity or base was flat or slightly convex and its outline was a curvilinear triangle. Anteriorly, the cavity narrowed to a tapering point or rounded tip. The general shape was that of a triangular pyramid with rounded angles. In one case the rostrocaudal orientation was inverted—that is, the base was rostral and the apex caudal. The smaller cavities were oval.

The lining of the cavities was whitish and smooth and to the naked eye resembled ependyma. Around the cavity there often was an ill-defined gelatinous area of greyish, slightly translucent tissue, softer in consistency than the surrounding white matter. The cavities contained a small amount of clear, colourless fluid.

Microscopically the lining of the accessory cavities was unmistakably ependyma of relatively low-columnar type. In a few cases there were areas where the ependyma had desquamated. The subependymal tissue was made up of a loose, gliofibrillar mesh, interspersed with numerous small groups of aberrant ependymal cells. Some of these groups formed small true rosettes, with a central cavity. The loose subependymal glia, which corresponded to the gross gelatinous layer, merged imperceptibly with the more closely meshed surrounding feltwork of glia and medullated nerve fibres. In the subependymal glia there often were amyloid bodies, similar to those found near the ventricles in many adult and senile brains.

Examination of the tissue between the tip of the occipital horn and the accessory cavity revealed a constant structure whose presence would seem to be of value in explaining the origin of the cavities as well as, to some extent, the variability of the human occipital horn itself. Joining the tip of the ventricle to the cavity was a narrow streak of grey tissue, somewhat softer than the surrounding white matter. In colour and consistency, this streak resembled the tissue about the accessory cavities and subependymal tissue generally. Microscopically, it was a band measuring from 30μ to 200μ wide, made up of glial mesh, looser than in surrounding areas and markedly richer in macroglia, while it was poorer in oligodendroglia. Amyloid bodies were often present. Myelin fibres were absent or very rare in the streak, and there was a sharp demarcation between the neighbouring white matter and the non-medullated band. In and near the streak were large veins, all running approximately parallel to it. In the middle of the streak most of the sections showed a single or a double row of cuboidal ependymal cells. Here and there the cell rows gave place to one or more true rosettes of ependymal cells, with a central, round, or oval empty cavity of variable

size, from 20μ to 100μ in diameter. In some places were seen clusters of from two to eight minute rosettes side by side.

The entire structure joining the tip of the ventricle to the accessory cavity, or other homologous structures, will hereafter be denoted the "ependymal streak." A cavity lined with ependyma but distinct from the occipital horn will be called an "accessory occipital cavity," whether it be seen by the naked eye or microscopically.

The majority of adult brains with gross accessory cavities presented a second ependymal streak, dorsal to the first, shorter than it, and not terminated by a gross cavity. In all other respects—the peculiar texture, the rows of ependymal cells, the parallel blood-vessels, the rosettes—the dorsal and the ventral streaks were similar. In some cases, the dorsal streak ended in a microscopical ependymal cavity. The topographical relations of the double ependymal streak can best be understood by considering the form of the tip of the occipital horn. In most of the occipital lobes with gross accessory cavities, the horn was of fishtail type, i.e. it was flattened from side to side and divided into two terminal portions, which may be termed the dorsal and the ventral recess respectively, separated from one another by a fullness in the retroventricular tissue. This disposition is determined by the convergence of four surfaces of different curvatures, different both in sign and degree. The four surfaces are : (1) medially, the calcar avis, always strongly convex ; (2) dorsally, the bulb of the forceps major, smaller than the calcar and always forming a flatter bulge or even a plane surface ; (3) laterally, the tapetum, whose ventricular surface is concave, but far less curved than the calcar ; (4) ventrally, the narrow collateral eminence, either flat or slightly convex. While it is customary to describe the cross-section of the occipital horn as triangular, it is more often quadrilateral at least in its rostral half. The general form of the cavity resulting from this arrangement is that of a vertical concavo-convex lens, with a strongly curved concave medial surface and a slightly curved convex lateral surface. As one follows the occipital horn caudalward on successive coronal sections, one usually sees the floor and the roof flatten out, if they were not already flat at the start. The floor often becomes slightly concave. Its curve is then continuous with that of the lateral wall, but the curvature is greater, so that the combined lateroventral wall is shaped like a capital letter J with the upright line slightly curved. In any case, the floor and the roof bulge less and less caudalward. At the same time, the medial and lateral walls join at an acute angle. The apex of the calcarine bulge first meets the tapetum, thus subdividing the occipital horn into its dorsal and ventral recesses. Further caudalward, the zone of coalescence of the medial and lateral walls increases in dorsoventral extent, the two recesses narrow and typically end both in a point. The dorsal recess is almost always triangular in cross-section, the base of the triangle being the roof of the ventricle and the apex the meeting-point of the calcar and the tapetum. The ventral recess is triangular when the floor of the ventricle remains convex or flat. In the frequent cases where the caudal portion of the floor is concave, the ventral recess is crescent-shaped, lying between the ventricular floor and the ventral aspect of the calcar. The ventral recess is always longer than the dorsal.

On a coronal section immediately caudal to the tip of the occipital horn, one sees a narrow grey area which has all the gross and microscopical characters of the ependymal streaks and whose form is an exact projection of the caudal extremity of the ventricle with its subependymal tissue. The relations of this structure are the same as those of the tip of the occipital horn. A few millimetres further caudalward, the retroventricular ependymal mass divides into a dorsal and a ventral portion, which continue as the two separate streaks.

Of the 34 brains without gross accessory cavities, seven came from individuals aged 19 months or less and contained no ependymal streaks. Of the remaining 27, coming from individuals aged 14 to 84 years, all but two presented ependymal streaks caudal to the tip of the occipital horns. In microscopical sections small rosettes were

often seen in these streaks. Complete serial sections of these areas could not be undertaken because of material limitations. It is possible that rosettes would have been found constantly in the ependymal streaks. In any case, it is evident that the adult brains with and most of those without gross accessory cavities share the same fundamental structure, the ependymal streak, which, like ependymal tissue everywhere and even in the disordered growth of neoplasms, tends to form epithelium-lined cavities. In some cases, the cavities are large enough to be seen with the naked eye ; in other cases they are microscopical.

The tip of the occipital horn, its recesses, and the ependymal streaks present remarkable variations in the adult brains. Aside from the frequent bifid or fish-tail form which I have taken as the type, there are forms in which only one recess, ventral to the calcar, is present. Whether there be one or two recesses, the ventral recess may be very long and narrow. Its cross-section may be rounded, corresponding to the classical digitiform type, or it may be triangular, crescent-shaped, or reduced to a laterally flattened potential cleft. It may broaden terminally into a bulb, which is then the homologue of a gross accessory cavity, the intervening long ventral recess corresponding to a ventral ependymal streak. In some cases the callosal bulb is oriented so as to belong more nearly to the medial wall than to the roof of the ventricle. It is in these cases that the cross-section of the occipital horn approximates a triangle. If, then, the curve of the bulb continues that of the calcar, the dorsal recess is absent or barely indicated, while the occipital horn as a whole tapers to a point corresponding to the ventral recess, which may itself be short or long. Intermediate between this type and the fish-tail is a triangular cavity tapering rapidly to a point ventral to the calcar, but with a slight dorsal depression between the calcar and the bulb. A small dorsal or ventral recess may merge into a long ependymal streak. In the classification of all the adult occipital horns into long and short types, those with long ventral recesses, continuing beyond the rest of the ventricle, were termed long. The bifid type with the ventral recess ending practically at the same level as the rest of the ventricle and the rapidly tapering triangular type were termed short.

In the brains of the five full-term and 8-month babies in the series, in the one 2 months old, and in the one 19 months old the occipital horns were broad, round in cross-section, and ending in a round tip. Of the various eminences characteristic of the region, only the calcar avis was present. Its bulge was far less sharply marked off from the neighbouring portions of the ventricular walls than in the adult. It diminished gradually caudalward until, in a section just rostral to the tip of the ventricle, the calcar was only represented by a slight flattening of the concave medial wall or was entirely absent. Caudal to the tip of the ventricle, no ependymal streaks could be found, either with the naked eye or microscopically. In one baby of 6 months the occipital horn on one side ended in a typical fish-tail with ependymal streaks and a gross accessory cavity.

Discussion

All of these data suggest a common explanation—the existence in the human embryo of a primary, broad, rounded occipital horn, ending in a round tip. Following the growth of the various structures which indent the occipital horn—calcar avis, callosal bulb, tapetum, collateral eminence, optic radiations—part of the ventricular cavity is obliterated by apposition and concrescence of its walls. This obliteration takes place at an extrauterine period of development, when the ependyma is completely differentiated. Thus, unlike what happens in the early embryonic obliteration of the dorsal portion of the central canal of the spinal cord, the ependyma in the occipital lobe persists as a solid sheet of variable extent between contiguous layers of white matter. In this sheet of ependyma are small cavities, microscopical or visible to the naked eye, which may be either closed-off remnants of the ventricular cavity or else newly formed rosettes in a one-time solid ependymal streak.

This hypothesis would well explain, as far as it goes, the great variability of the occipital horn in different individuals and the differences between right and left occipital horns in the same individual. Just why the rate of growth of the structure surrounding the occipital horn should be so variable requires an explanation which only further study can supply.

A second hypothesis is that of a cavity following some destructive lesion and secondarily lined with ependyma. There is nothing in favour of this hypothesis. Indeed, cavities accompanying ischæmic malacia, hæmorrhage, compression by tumours, suppuration, etc., never acquire an ependymal lining. Furthermore, it would seem improbable that such cavities should occur so frequently in the same situation. In any case, this hypothesis could not account for the ependymal streaks.

The ependymal streaks and the accessory occipital cavities mark the boundary between important medullated fibre tracts—the bulb and the principal bundle of the forceps major, the tapetum, the stratum calcarinum—and should be able to serve as added landmarks in distinguishing these tracts in the study of secondary degenerations. The ependymal structures are easily recognizable microscopically with different stains, and as a rule, can be seen with the unaided eye.

The number of cases observed (16 in an unselected series of 50 cases) is as yet too small to permit of any statistically valid conclusions with respect to sex and race incidence and the like. It would seem that the presence of gross accessory occipital cavities is not rare. As no constant lesion or malformation is associated with this condition, it must be considered to be one of the many anatomical variants of the occipital horn.

A fact which may be significant is the greater frequency of the short type of occipital horn and of gross accessory cavities on the right side than on the left. According to Engel (quoted from Testut and Latarjet (1929)), the left occipital horn is longer than the right in two-thirds of the cases. In only one case of my series was a unilateral cavity present in the left occipital lobe. It would have been interesting to know whether this individual was left-handed. Unfortunately there was no record of handedness in any of the cases studied. Attempts at seeking anatomical correlates of handedness would be greatly facilitated if all hospitals emulated the simple practice of the Salpêtrière in Paris, where, following the instructions of Professor Georges Guillain, the handedness of each patient is noted on admission.

A final problem suggested by the accessory cavities is the origin of the fluid contained in them. The same problem is presented by the true rosettes in ependymal tumours. No choroid epithelium was observed in any of the accessory occipital cavities. One must perforce suppose either that the fluid reaches the cavity by transudation from the surrounding tissue spaces or that the ependyma of the cavities is endowed with secretory functions, such as Wislocki and Putnam (1924) demonstrated in the area postrema of the fourth ventricle. In either case it is difficult to imagine any adequate mechanism of absorption of the contained fluid. The ependyma is not flattened, as it would be if subjected to prolonged pressure. It must therefore be supposed either that the fluid is produced slowly enough to be removed by the slight absorptive capacity of the ependyma itself or else that after the fluid fills the cavity no further production of fluid takes place.

Summary

Small cavities caudal to the tip of the posterior portion of the lateral ventricle were observed in one or both occipital lobes in 16 out of 50 unselected human brains, more frequently in the right than in the left hemisphere. Microscopically these cavities were lined with ependyma.

Between the cavity and the tip of the occipital horn was a grey sheet or streak, shaped in cross-section like a projection of the end of the ventricle. Microscopically

this streak was a single or a double row of ependymal cells flanked by tissue of subependymal type, with blood vessels parallel to the ependyma. In places the solid ependymal layer was replaced by ependyma-lined microscopic cavities which were homologous to the gross cavities.

In many occipital lobes without gross cavities an ependymal streak and microscopic cavities were found.

In new-born babies and rarely in adults the occipital horn was rounded and there was no ependymal streak or accessory cavity.

In most adults the occipital horn was flattened by the calcar avis and the bulb of the forceps major, suggesting that in extrauterine life the tip of the occipital horn is occluded to a variable extent by the growth of the surrounding tissues, thus giving rise to the ependymal streak. This hypothesis would also explain the great variability of size and conformation of the adult occipital horn.

My indebtedness is due to Dr. James R. Lisa, Pathologist at the City Hospital, for placing the material and the records at my disposal.

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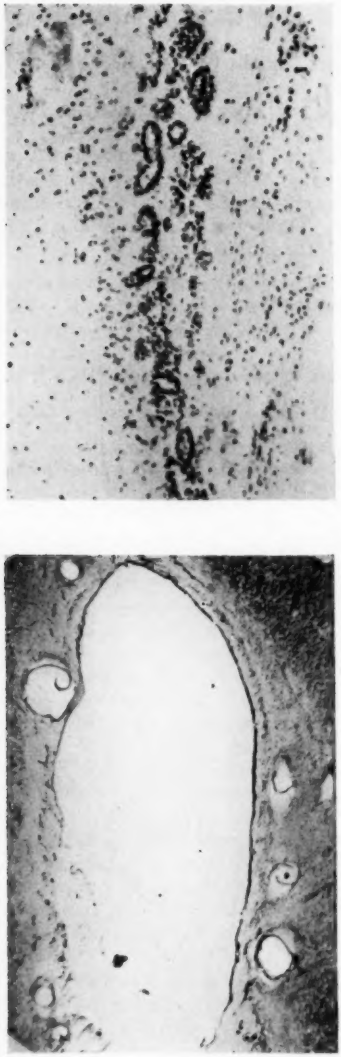
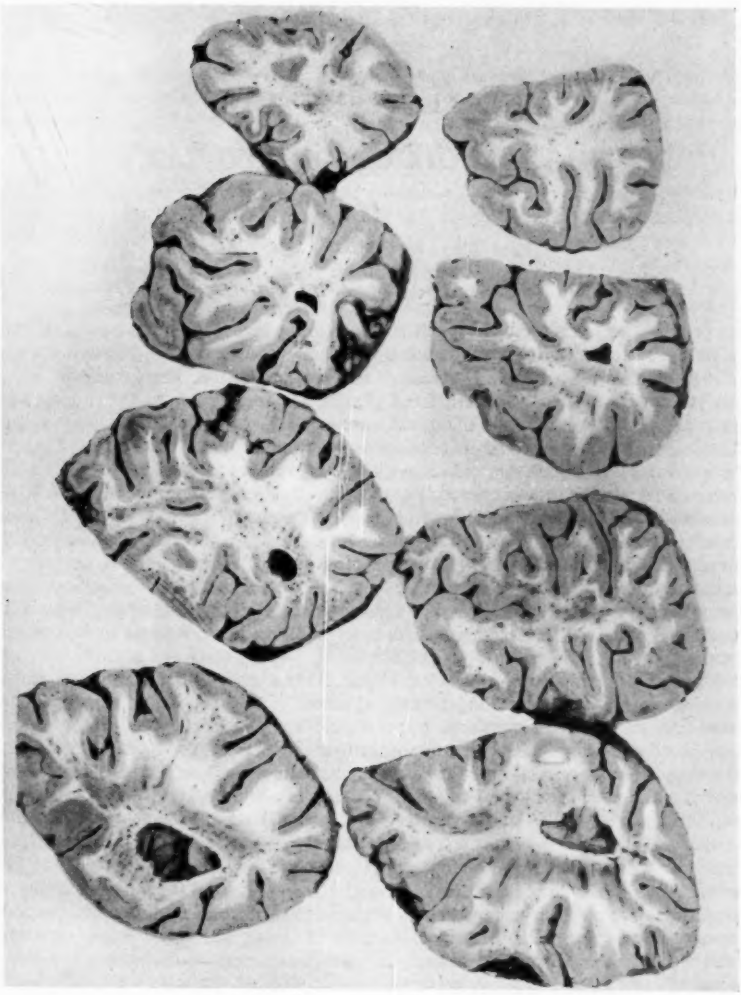


Fig. 1.—*Bottom :* an ependymal streak at 14 mm. caudal to the ventricular tip, showing solid epithelial rows and multiple rosettes. This case had no gross accessory cavities. Sagittal ; ×150. *Top :* an accessory occipital cavity from a different case. Transverse. The ependyma (dark layer) is desquamated over about one-third of the cavity. The thin-walled blood vessels in the subependymal tissue are all cut transversely, i.e. they are parallel to the direction of the ependymal streak ; ×20.

CAUDAL



LEFT

RIGHT

ROSTRAL

Fig. 2.—Rostral aspect of successive coronal slabs of the occipital lobes of an adult human brain. The left occipital lobe has a long and undifferentiated type of posterior horn, ending in a rounded tip. The right horn is of the short fishtail type, indented by the bulb, the calcar, the collateral eminence, and, in this case, secondary eminences. Note the two recesses, of which the ventral is deeper than the dorsal. The second slab shows the grey ependymal streak, between the stratum calcarinum medially and, laterally, the tapetum and optic radiations. The third section shows the caudal segment of the accessory cavity. All ependymal structures are absent from the fourth section.

CRITICAL REVIEW

THE PATHOLOGY OF APOPLEXY

BY

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THE problem of the apoplectic insult presents difficulties which are typical of those met in all investigations in pathological anatomy. While the apoplexy itself is one of the most striking and dramatic events in clinical medicine, the anatomist has to draw his conclusions from a static fixed picture and to reconstruct the process in all its stages from the comparison of a great number of different final states. No wonder that the interpretations are never conformable and reflect the historical development of physiological and pathological knowledge. It is still more surprising that, as shall be seen, not only the interpretations but even the facts of observation differ so largely with different investigators. This incongruity is caused to a certain extent by a confusion in terminology, so that different authors obviously apply the same term for different things or different terms for the same thing. Therefore it is necessary to summarize briefly what is more or less generally accepted as the anatomical equivalent of apoplexy. Three different kinds of lesions can be sharply distinguished. First, there is the massive hæmorrhagic apoplexy in which is found a more or less massive hæmorrhage in one or several areas of the brain. Often from the area in which the hæmorrhage occurs a solid clot is easily separated and falls out on section, leaving behind a cavity with hæmorrhagic walls. Secondly, there is the softening, an area which does not show hæmorrhage but a diminished consistency and slight or more pronounced shrinking or discoloration of the tissue. Thirdly, there is the hæmorrhagic infarction, which presents a picture similar to the softening and distinguished from it only by the extravasation of blood into the softened area, usually in innumerable small stipples. A great deal of confusion in the more recent literature is due to the fact that a distinction is not always clearly drawn between the first and the last types, namely, the apoplectic hæmorrhage and the hæmorrhagic infarction. As at least one of these three types of lesion is the usual finding in cases which clinically present the picture of apoplexy, we may regard them as the anatomical basis of this process. However, in a large number of such cases one finds other additional lesions, small foci of necrosis and scar formation in the basal ganglia *état criblé*, *état lacunaire*), and in the cortex. As these smaller areas of necrosis differ from the large areas of softening only in size, it seems to be obvious that they represent the anatomical equivalent of short transitory attacks of fainting, giddiness, aphasia, blindness, or paresis which may occur in those patients.

In addition to the three chief types, one ought to mention certain cases which Westphal (1926) has brought into prominence. These were cases which showed the typical clinical picture of apoplexy but failed to show any anatomical lesion. As a microscopical examination in these cases was not done or was incomplete, it is possible that these cases presented only early stages of necrosis which are macroscopically undiscovered and would be revealed on exact histological examination. Therefore these doubtful cases may be eliminated from our consideration.

Vessel Lesions as the Causes of Cerebral Hæmorrhage in Arteriosclerosis and Hypertension

In reading the description of cases, it is quite evident that most of the anatomical investigations on apoplexy deal with the first-mentioned type, the apoplectic massive hæmorrhage. Until a few decades ago the pathogenesis of this disturbance seemed to be clear. Charcot's view was more or less accepted. Charcot and Bouchard (1868) had reported 77 cases of cerebral hæmorrhage, in all of which they found circumscribed small swellings of the arterial branches within the brain. These miliary aneurysms were 0·2 to 1 mm. in size and had a globular or fusiform shape. Their number varied from 2 to 100 in each case. As Charcot and Bouchard saw aneurysms in the wall of the hæmorrhage or in close relationship to the hæmorrhagic area or, less frequently, somewhere in the brain and in no direct relationship to the focus of hæmorrhage, it seemed probable that the hæmorrhage was associated with these aneurysms. In order to prove that these aneurysms are really the source of hæmorrhage the actual rupture must be seen. It is therefore important to note that in only three of their cases did Charcot and Bouchard mention definite ruptures of aneurysms.

During the following years (Zenker, 1872 ; Arndt, 1878 ; Eichler, 1878 ; Rindfleisch, 1878 ; Turner, 1882 ; Monakow, 1905) Charcot's theory was accepted with certain alterations, chiefly concerning the histological nature of the aneurysms. The causal connection between the aneurysms and the hæmorrhage seemed to be evident. Löwenfeld (1886) pointed out in a thorough investigation of 17 cases of hæmorrhagic apoplexy with recent and old areas of hæmorrhage that the hæmorrhage did not necessarily arise from miliary aneurysms. He found severe changes, " simple, fatty, and granular degeneration " in the walls of the cerebral vessels in such cases. These changes, however, did not occur only within the area of hæmorrhage, although they were more marked and more frequent there than in other parts of the brain. One of his cases did not show any ruptured miliary aneurysm, but a ruptured vessel without a miliary aneurysm. In Löwenfeld's cases the rupture of the vessel wall and the mechanical injury of the brain substance by the violence of the released blood stream, thereby producing secondary ruptures of the surrounding vessels, were sufficient to explain the picture.

After Eppinger's (1887) general description and classification of aneurysms, the most thorough and exact investigation into the nature of these special aneurysmal formations was done by Pick (1910) and his collaborator Ellis (1909). They showed that these formations were not true aneurysms but either subadventitial hæmorrhages or extramural hæmatomas surrounded by fibrin, destroyed brain substance, and elements of the vessel wall itself. They are produced by the rupture of a previously normal vessel or of a vessel which has already shown dissection of its layers. According to Pick and Ellis a fatal hæmorrhage can never occur from these miliary aneurysms, and the aneurysms which actually are seen to be ruptured within the hæmorrhagic area are of a larger size than the miliary aneurysms of Charcot and Bouchard. Pick also emphasized that the tissue surrounding the miliary aneurysms never showed a marked cellular reaction ; thus it may be presumed that they did not arise a long time before death. Shennan (1915) came to a similar conclusion, namely, that a local dilatation of the diseased vessel ruptures and produces the apoplectic hæmorrhage. This local dilatation is not a pre-existent chronic change, but immediately precedes the rupture. It is also of a larger size than Charcot's miliary aneurysm. The hæmorrhage may follow the formation of a dissecting aneurysm similar to the dissecting aneurysms of the large arterial trunks. Shennan states that he found in all his cases only one single miliary aneurysm which corresponded to Charcot's and Bouchard's description. Stein (1895) had previously reported six cases of apoplexy in which an exact examination had not shown any miliary aneurysms. In some of

the vessels the adventitial space was filled with blood or detritus. Thus these findings showed even more conclusively that the classical theory of the causal significance of the miliary aneurysms in apoplectic hæmorrhage was not entirely adequate.

This was the situation when the paper by Rosenblath (1918) appeared. It formed the basis of discussion in most of the succeeding investigations, just as Charcot's and Bouchard's work had done. Rosenblath found in an exact macroscopical and histological study of 11 cases, of 2 hours' to 15 days' duration between apoplexy and death, that the hæmorrhage never occurred from one vessel but from many vessels simultaneously, that vessels and brain substance had both undergone acute necrosis in the area of hæmorrhage, and that the form of the lesion and the mode of expansion of the hæmorrhage could not be explained by the mechanical violence of the blood stream destroying the tissue. Hence he concluded that the necrosis of vessel and brain tissue was the primary event and the hæmorrhage followed into a previously necrotic area. His hypothetical explanation of this process is unlikely. He postulated that a toxic ferment produced by the contracted kidney caused the acute necrosis in the brain. This hypothesis was not accepted by succeeding investigators. Although the whole conception was a great stimulus to investigators who followed him, some of his results appear open to criticism. For instance, the presence of many punctate hæmorrhages in the periphery is no evidence that the entire lesion must have been produced by the confluence of such small hæmorrhages. The violence of a massive hæmorrhage can undoubtedly produce small punctate hæmorrhages in the neighbourhood simply by the mechanical damage to the small vessels. Similar small hæmorrhages may be found round a fresh bullet-hole in the brain (Stämmler, 1927) or round an experimentally injected clot of chicken blood in a dog's brain (Rühl, 1927). But there seems to be a difference between these secondarily produced marginal small hæmorrhages and the small foci of hæmorrhage which are to be seen in the peripheral zone of gross apoplectic lesions. This point requires further investigation. Rosenblath argues that the form of the lesions in apoplectic hæmorrhages is very much against the view that they are produced by a bursting vessel and the mechanical destruction of the surrounding brain tissue. Actually the egg-shape of a certain type of putamen-claustrum hæmorrhage is difficult to explain by the mere mechanical force of the outflow of blood. However, in the marginal zone of other apoplectic hæmorrhages, especially in the lower brain stem, one can show that the hæmorrhage does in fact follow the planes of least resistance in the fibre network (Stern, 1935). Rosenblath maintains that the smooth walls of the hæmorrhagic cavity and the hæmorrhages which stop short immediately underneath the ventricular wall are much against the mechanical theory. Some hæmorrhages show the form of slits ; there are cases in which the walls of the cavity show torn and irregular tissue (Böhne, 1927) and, as is well known, there are many cases in which the hæmorrhage actually breaks through into the ventricle.

Several important points in Rosenblath's theory have received ample proof. The source of hæmorrhage is multiple in most cases. The vessels in the entire area of hæmorrhage undergo " arterionecrosis." The expansion of the hæmorrhage cannot be explained by rupture and mechanical violence only. Lastly, the small miliary dissecting aneurysms of Charcot and Bouchard are not the cause but one of the accidental results of the process.

Lindemann (1924) confirms that there are always many vessels from which the hæmorrhage occurs and that the hæmorrhage is not so much produced by rupture as by diapedesis and " diaresis." Böhne (1927) compared the volume of both hemispheres, the hæmorrhagic and the normal hemisphere of the same brain. The volume of the apoplectic hemisphere may be a quarter more than that of the healthy one. Thus there must be a considerable displacement of brain substance by the hæmorrhage. Rosenblath had emphasized that in many cases one cannot judge by external appearances in which part of the brain the hæmorrhage had occurred and that the hæmorrhage did not obviously displace a considerable amount of brain

substance. On the other hand, Böhne's contrary statement is not against the presumption of a preceding necrosis.

Transient Disturbances of the Vessels as the Cause of Hæmorrhage and Softening

Westphal and Bär (1926) confirmed Rosenblath's findings, but tried to replace his hypothesis of a chemical noxis destroying the brain tissue by another which seemed more in accordance with clinical observations. Westphal's view is based on certain facts. Spasm of the retinal vessels has been observed occasionally in patients with arterial hypertension and in whom certain paradoxical microcapillary reactions have been observed. There was no " reactive hyperæmia," but rather angiospasm after arterial compression. Moreover, it is true that apoplexy is often preceded by minor fits which obviously do not lead to a gross hæmorrhage. From these facts, combined with the anatomical findings, Westphal draws the conclusion that in arterial hypertension a local arterial spasm producing ischæmia and necrosis preceded the hæmorrhage. This hypothesis necessitates the assumption that the cause of the necrosis of the artery is a preceding arteriospasm, and that hæmorrhage follows the arterionecrosis with subsequent tissue necrosis. Westphal mentions cases in which the clinical picture was that of a typical stroke with following paresis in a patient with arterial hypertension and in which at post mortem neither a lesion in the brain nor an obstruction of the vessels was found. In such cases a transitory ischæmia due to an arterial spasm may have produced the stroke. We have already mentioned that these cases are, at least as far as the negative findings in the brain are concerned, doubtful. Jaffé (1927) studied the vessels in the case of a gross cerebral hæmorrhage occurring in an eclamptic woman of 33 years who had a high arterial pressure and no signs of arteriosclerosis. He followed Westphal's theory. Globus and Strauss (1929) found in brains with apoplectic hæmorrhage signs of previous circulatory damage, such as scars, more recent softenings, and general gliosis. From these findings they drew the conclusion that the degeneration of the surrounding brain tissue as well as vessel disease and hypertension is an important factor in the production of hæmorrhages. They even mentioned one case which " in the absence of a pre-existing cerebral softening escaped spontaneous massive hæmorrhage " though diffuse chronic disease of the cerebral vessels associated with hypertension was present. As in this case no description of the microscopical findings was given, circulatory disturbances can hardly be excluded. But even the pre-existence of disintegration of the cerebral tissue does not prove that it is a necessary condition in the production of hæmorrhage. We can only say that the same diseases which tend to produce softening and diffuse proliferative changes in interstitial tissues also lead to hæmorrhages. The immediately causal connection between the two processes postulated by Westphal is not proved.

Westphal's view is fairly typical of current opinion regarding our present subject. While in the classical literature hæmorrhage was almost identical with vessel rupture and softening with thrombosis, the conception of transient " functional " vascular disturbances has dominated recent work.

It is, however, often very difficult to differentiate clearly between unfounded conjectures and fruitful theories. Therefore it is necessary to enquire why the basis of a direct causal connection between a visible organic lesion of a vessel and the resulting brain lesion was abandoned in favour of much vaguer and less-defined conceptions. It is quite a common experience in morbid anatomy that there may be no immedate connection between the degree of arteriosclerosis on one side and the amount or number of hæmorrhages and softenings on the other. One may find on the one hand extensive and numerous lesions in a brain with only slight disease of the vessels, and on the other no gross lesions in a brain with severe arteriosclerosis (Neubürger, 1930). If a comparison is made in arteriosclerotic brains between the extracerebral vessels in an area of high " vulnerability," such as the pons, and those of a very " resistant " area near to it, as the hypothalamus (Stern, 1935), the degree

of arteriosclerosis is found to be the same in both. Such observations deal only with a gross examination of extracerebral vessels. In regard to the intracerebral vessels, the occurrence of cerebral necrosis in the absence of visible obstruction is reported by a number of different observers. Foix and Hillemand (1924) report cases of cerebral softening in which serial sections of the arteries to the softened areas did not reveal the presence of either thrombosis or obliteration. Unfortunately these cases are only mentioned without details in a discussion. Spielmeyer (1924) reported cases of arteriosclerosis with necrotic areas of apparently the same age scattered all over the cortex in which the arteries supplying these areas showed no recognizable change. He speaks of " vasomotor " disturbances in cerebral arteriosclerosis. Neubürger (1926) reports an interesting case of a healthy man of 31 who, after a traumatic commotion with a lucid interval of 1 hour, developed a hemiplegia and died 2 days later. At post mortem no lesion of the skull was found ; there was a softening in the area of one of the branches of the middle cerebral artery though the vessels were healthy and patent. Neubürger draws a parallel to the post-traumatic segmental arterial spasm which has been observed in living patients by surgeons (Küttner and Baruch, 1920 ; Vianney, 1919). In most of these surgical cases, however, a lesion of a neighbouring vein seems to have preceded the spasm of the artery. A good example is the case of Duval, Lhermitte, and Vermes (1935), in which the ligature of a jugular vein was followed by a hæmorrhagic softening on the same side. In 1927 Rosenblath described 12 cases of arterial hypertension with cerebral softenings in which he found vascular changes similar to the arterio-capillary fibrosis of Gull and Sutton (1872). But as these vascular changes were not merely confined to the areas of softening it is probable that such vascular changes were not the immediate cause of the necrosis. No other obstruction of the arteries, embolic or thrombotic, was seen. H. Spatz (1935) described a case of the cerebral form of Buerger's disease (thrombo-angiitis obliterans) in a man of 43 years. There were varying symptoms of speech disorder, mental disturbance, and transitory pareses throughout the 7 years of the illness. The anatomical examination finally revealed a proliferative endarteritis and organizing thrombosis of both carotid arteries and several of their branches, especially the left middle cerebral artery, which resulted in multiple diffuse lesions of the nervous tissue. There were similar changes in the coronary arteries. There was no evidence of true arteriosclerosis. In the clinical history there was nothing suggestive of any gangrenous disease of the limbs, though there was considerable evidence of frequent vasomotor disturbance of the extremities, such as paroxysms of coldness and lividity of hands and feet. The blood pressure was normal. From this and from the way in which the neurological and mental symptoms developed, Spatz concluded that cerebral angiospasm had preceded the organic disease of the vessels. If this was so we still do not know if the cerebral softenings did occur at a stage when the vascular disease was of a purely " functional " character. Guillain and Bertrand (1932) demonstrated a case with intact arteries in which there were symmetrical necroses of long standing in the parietal and occipital lobes. Riser (1935), in a case of cerebral softening, injected with gelatine the arteries supplying the softened area. The arteries showed some small narrowings produced by arteriosclerotic plaques, but they were otherwise patent.

From all these facts it is quite evident that necrotic lesions of the brain may occur without any associated visible obstruction (thrombosis, embolism, obliteration) of the arteries. As the shape and distribution of such lesions suggest their arterial origin, they must have resulted from transient " functional," " non-organic," or " vasomotor " disturbances. Thus for the pathogenesis of a great number of cerebral softenings the function of the vessels becomes more important than their morphological picture. This view receives further support from those cases where completely obstructed arteries have not caused any necrosis (Pagnicz, 1902 ; Claude and Cuel, 1927 ; Ley, 1931) ; as is well known, a slow development of the obliterative process may allow of a sufficient anastomotic supply.

The next important problem is to determine if the same or similar transient " functional " disturbances of the vessels, the nature of which is not known, can produce not only softenings but also hæmorrhages. Lampert and Müller (1925) have carried out an interesting experiment regarding the question of rupture of vessels by an increase of intra-arterial pressure. In human bodies of subjects who died with cerebral arteriosclerosis they injected fluid under a high pressure into both carotids. Only by applying pressure of 1 to 2½ atmospheres for 6 minutes did they succeed in producing ruptures with destruction of brain substance by the leaking fluid. Such pressures never occur in human pathology. From the results of such experiments and from the histological findings of Rosenblath (1918, 1926), Lindemann (1924), Pollak and Rezek (1927), Alajouanine, Thurel, and Hornet (1936), it may be said that cerebral hæmorrhage is not likely to be the result of rupture of one or a few rigid blood vessels from a sudden increase in blood pressure.

There is no supporting fact of observation either in the brain or in any other organ for Westphal's theory of an angiospasm causing not only necrosis of the parenchyma but also necrosis of the " spastic " vessel itself. Let us eliminate the massive apoplectic hæmorrhage and consider only the hæmorrhagic infarction. By the latter is meant the necrobiotic change with intact continuity of tissue and numerous capillary hæmorrhages within the damaged area. Ricker (1924) has shown in experiments on the pancreas and ear of the rabbit that necrosis does not actually occur during the period of angiospasm and ischæmia. It occurs during the stage of prestasis when the blood stream in the precapillary and capillary bed is extremely slowed or when the blood flow has stopped and the vessels are engorged. Ricker shows that if he applied to an artery a stimulus of varying character the result to be observed in the precapillaries and the capillaries belonging to that artery was always the same. With a light stimulus angiospasm and ischæmia occurred. If he increased the strength of the stimulus the arterioles and capillaries widened and the leucocytes took up a marginal position and emigrated into the tissue through the stomata of the capillary wall ; erythrodiapedesis then took place. At this stage the earliest necrotic changes of the tissue were observed. If he increased the stimulus still further stasis occurred with massive erythrodiapedesis. Ricker observed that these processes occurred constantly in the same order and that there was no difference if he applied chemical, mechanical, or thermal stimuli. He assumes that the mechanism producing these changes is nervous. It is not possible here to go into any further details of the physiology of the circulation ; but even if Ricker's findings really represent an invariable " law," there are reasons enough to believe that these observations cannot immediately be applied to the human brain. With this reservation, it may nevertheless be said that Ricker's attempted explanation best meets the histological picture of ischæmic and hæmorrhagic infarctions. In infarction of the brain it is possible to see all the different stages described by Ricker in his experiments. The ganglion cells may have undergone necrobiotic changes, the capillaries being extremely engorged ; or the ganglion cells and capillaries may show the same changes with the leucocytes scattered through the necrotic tissue ; or the ganglion cells may be unstainable by basic aniline dyes and there may be massive erythrodiapedesis ; or there may be erythro- and leucodiapedesis in a tissue in which the damaged ganglion cells are still to be seen. In other words, all stages described by Ricker in the living animal may be seen in one or other lesion found at post mortem of the human.

The necrosis in typical infarcations may be confined to a certain part of the grey matter, namely, the cortex or only the lower layers of the cortex. This sharp limitation cannot be explained by the angio-architecture, because some of the pial arteries pass through the cortex into the deep parts of the white matter. It could be explained by the higher vulnerability of the nerve cells to ischæmic noxa, a point which is so often emphasized. But the fact that the hæmorrhage in hæmorrhagic infarction is almost exactly confined to the area where the necroses occur is hardly explained in this way. If Ricker is correct in stating that the disturbance in the " terminal " vascular

area—prestasis, leucodiapedesis, erythrodiapedesis, stasis, necrosis—is due to a stimulus to the arterial trunk which is transmitted to the periphery by a nervous mechanism, it is necessary to prove that such a pathological stimulus to the arterial wall does occur. Consideration of this leads to a discussion of Schwartz's (1930) theory of the embolic lesions. Schwartz considers that the obstructive factor in embolism is not the deciding factor, because it does not explain the limitation of the small hæmorrhages to certain parts of the grey matter such as are quite common in embolic infarction. He believes that the true cause is the trauma to the arterial wall by the sudden lodgement of the embolus. This lodgement brings about a functional disturbance in the terminal distribution of the artery. This disturbance follows the same mechanism Ricker has described in his experiments. Schwartz's extensive monograph on the pathology of apoplexy is based on Ricker's theory. The result is that all lesions in arteriosclerosis, embolism, and arterial hypertension, with the exceptions of massive thrombotic obstructions in arteriosclerosis, are due to pathological stimuli to the arterial trunk which cause by a nervous mechanism circulatory disturbance in the terminal distribution. Against such an hypothesis it must once more be emphasized that the circulation in the brain is in many ways different from that in the organs Ricker has studied, and, moreover, the analogy to Ricker's different stages is only to be found in ischæmic and hæmorrhagic infarctions and not in massive apoplectic hæmorrhages such as are so common in the basal ganglia, the white matter of the cerebrum, and in the pons. The material which Schwartz demonstrates illustrates once more the inadequacy of the classical mechanical theories. His hypothesis, however, is insufficiently supported by physiological facts. The bilateral and absolutely symmetrical softenings and small hæmorrhages in the basal ganglia which he shows may possibly be explained by a nervous impulse transmitted symmetrically along the arterial tree to its terminal branches on both sides. Physiological evidence in favour of this is, however, wanting. Again, these findings are not frequent enough to give sufficient basis for such a general explanation. Schwartz stresses the morphological similarity between embolic and hypertensive lesions to prove the similarity in pathogenesis—namely, a shock to the arterial trunk producing functional disturbance in the terminal vessels. It is known that embolus is likely to pass along vessels which continue the most direct stream of blood. As the lenticulo-striate arteries come off the middle cerebral artery opposite the end of the internal carotid, emboli passing up the carotid tend to lodge in the lenticulo-striate artery. This results in embolic lesions being found chiefly in the distribution of the lenticulo-striate arteries. As Schwartz found in arterial hypertension the site of hæmorrhage or softening to be in the distribution of the lenticulo-striate artery, he postulated that a sudden rise in arterial pressure passed as a wave from the heart along the most direct route—namely, into the lenticulo-striate artery. If this hypothesis be correct, it would be expected that those branches of the basilar artery which come off in a most direct line with the stream of blood would be the site of hæmorrhage and softening in cases of hypertension. The vessels so placed are those going to supply a part of the hypothalamus including the mamillary bodies. Vascular lesions in these areas are, however, extremely rare. In fact, it should be pointed out that the site of vascular lesions is commonly in the distribution of the pontine arteries, which come off the basilar artery at a right-angle or even more acutely.

Another attempt to explain the multiple hæmorrhages in areas of infarction has been put forward by Volhard (1932). He explained the hyperæmia as reactive to the " lack of oxygen " in the ischæmic area and the diapedic hæmorrhage only as a higher degree of this reactive hyperæmia.

It is only to be expected that Westphal's and Schwartz's hypothetical conceptions were followed by investigators who either returned completely to the classical and purely mechanical view or at least restricted themselves more to the observation of visible changes and rejected all other hypotheses (Rühl, 1927 ; Böhne, 1927 ; Stämmer, 1927 ; Green, 1930 ; Wolff, 1932 ; Hiller, 1935). Hiller states definitely from a

study of 59 cases that a state of disintegration (" status lacunaris," " status cribrosus " of Pierre Marie (1901)) is a prerequisite for the occurrence of cerebral hæmorrhage in arteriosclerosis and hypertension. He once more stresses the difference between the massive apoplectic hæmorrhage and the hæmorrhagic infarction. These have been confused so much in the polemics of recent years. In his cases the arteries of the distintegrated areas showed changes such as intimal proliferation, hyperplasia of the mesial coat, and dilatation of the perivascular space. The nervous tissue itself showed the well-known pictures of multiple softenings of different age. Therefore Hiller assumes as important for the pathogenesis of the hæmorrhage the changes of the arteries and the diminished perivascular resistance. He also emphasizes that a massive hæmorrhage with rupture into the ventricle is never caused by the confluence of numerous small diapedic hæmorrhages. It is very interesting that he found only very few hæmorrhagic apoplexies in nephritic arterial hypertension. It is also surprising and not in accordance with the findings of previous authors that in most of his arteriohypertensive patients under 60 years he found definite anatomical signs of arteriosclerosis. But even if—as Hiller states—the arteries in areas where the hæmorrhages occur show arteriosclerotic changes, the problem is essentially the same and has only changed its aspect. Because the arteriosclerosis, especially the hyperplastic change of the media of the small arteries, is probably the result of a functional overstrain (McCallum, 1936) and because early cases of arterial hypertension may show cerebral hæmorrhage without visible change in the arteries, it is logical to assume that the mechanism which leads to the vessel disease is also the cause of the final catastrophic event. Even if the marked arteriosclerosis in the arteries of the basal ganglia and pons were the constant cause of hæmorrhages in these areas, it remains to be determined why the arteriosclerosis is limited to these areas.

The Problem of Local Vulnerability

This leads to the question of the frequency of hæmorrhages and softenings in certain areas and their extreme rarity in others. If arterial hypertension affects the vessels of all parts of the central nervous system equally, why is it that the resulting lesions are not distributed according to mathematical probability ?

Ludlum (1909) studied the distribution of the gross apoplectic lesions under the immediate influence of Beevor's (1908) work. In the 91 cases reported the site of the lesion was as follows : there were seven hæmorrhages and six softenings in the area of the posterior cerebral artery, especially in the region of the thalamus ; six pontine lesions in the distribution of the basilar ; eight lesions in the area of distribution of the posterior communicating artery ; three lesions in that part of the internal capsule supplied by the anterior choroidal artery. All the remaining 61 lesions were in the distribution of the striate arteries. The distribution of the cerebral hæmorrhages is, according to W. S. Greenfield, as follows ; 75 per cent. occur in the region of the basal ganglia, 10–12 per cent. in the pons, 12 per cent. in the meninges, 1 per cent. or less in the cerebellum. Those occurring in relation to the meninges belong to a group not under discussion here. Boyd (1934) in his textbook gives a somewhat different distribution. In the order of frequency they are : (1) the area of the lenticulo-striate artery ; (2) the white matter of the frontal lobe (anterior cerebral artery) ; (3) the pons and the cerebellum. Schwartz (1930) emphasizes that the predilective areas are similar in apoplexy associated with arteriosclerosis, arterial hypertension, and embolism. There was only one exception among his observations : the cortex was more seldom affected in hypertension than in arteriosclerosis or embolism. The areas mostly affected are the middle third of the corpus striatum and the claustrum, then the thalamus and the pons. The globus pallidus and the medulla oblongata extremely rarely showed lesions. The most common cortical lesions in arteriosclerosis and embolism were located in the insula, the operculum, the supramarginal gyrus, and the superior temporal gyrus.

If it were possible to find a common feature in the vascular supply of all the vulnerable areas or in that of the most resistant parts of the central nervous system, it would undoubtedly advance knowledge as to the pathogenesis of these lesions. Duret (1874) and Heubner (1874) early stressed the importance of the presence of extracerebral arterial anastomoses in explaining the resistance of certain sites to vascular lesions. Kolisko (1891) thought that the comparatively rare occurrence of such lesions in the area of the anterior choroidal artery might be explained by the acute angle at which this vessel branches off the carotid artery. But it has already been mentioned that the pontine vessels branch acutely from the basilar artery and that hæmorrhages frequently occur from these branches. It is therefore unlikely that the angle of branching is closely associated with the cause of the lesions. The striate and pontine vessels have in common the fact that their diameters at their origin from the parent vessel and at their entrance into the brain substance are the same. These arteries have no extracerebral anastomoses. On the other hand, the extracerebral anastomoses so much stressed by the early authors are probably of importance when the anastomoses occur between vessels of the right and left side. Areas receiving such a double supply are extremely resistant to circulatory disturbance. As examples may be cited the spinal cord, colliculi, hypothalamic nuclei, and cortical areas adjoining the corpus callosum. The multiple ramifications of the cortical arteries, in contrast to the striate and pontine arteries, may cause the blood pressure to be " stepped down " gradually, as Boyd expresses it.

Regarding the intracerebral angioarchitecture of the areas most frequently affected, very little is known. Unfortunately Pfeiffer's (1928) method has not been applied to the human brain stem yet. Ludlum (1909) mentions that most of the infarctions formed in his cases occurred in the boundary zones of two different arteries. In the pons, areas which are supplied by one terminal artery are more often affected than those which are supplied by many small terminal arteries (Stern, 1935). Further investigations in this direction are necessary, more especially as Pfeiffer's method gives a more accurate picture of the intracerebral precapillary anastomoses.

As our knowledge of the embryological development of the cerebral vessels is still rudimentary, it is not possible to decide if the areas of high vulnerability represent areas in which minute developmental abnormalities more commonly occur. Oberndorfer (1928) recorded finding, in identical twins suffering from whooping-cough, hæmorrhages in almost the same place in the frontal lobe. In such cases microscopical developmental abnormalities of the vessels as described by Elkington (1935) may cause a local diminution of resistance to circulatory overstrain. In discussing local vulnerability mention should be made of the developmental anomalies in the walls of the cerebral vessels giving rise to " congenital " aneurysms (Fearnsides, 1916 ; Forbus, 1930). As these abnormalities are of greater importance in discussing the pathogenesis of subarachnoidal than of cerebral hæmorrhage, no further mention of them shall be made here. It is doubtful if the occurrence of congenital anomalies of the circle of Willis, such as hypoplasia or congenital obliteration of the posterior communicating artery found in arteriosclerotic brains with softenings and hæmorrhages are actually of as much pathogenetic importance as Saphir (1935) suggests. As these anomalies may frequently be found in otherwise healthy brains, the cases described by Saphir seem to represent a coincidence. In cases of hypertension occurring in acute, subacute, and chronic Bright's disease (Stern, 1935) the area of the basilar artery seems to be more often affected than other areas. This finding may be related to cerebral œdema or rapid increase of intracranial pressure, for hæmorrhages and softenings may be found in the branches of the basilar artery in brain tumours (Rosenhagen, 1932 ; Bannwarth, 1935), sunstroke (Stern, 1933), apoplectic ventricular hæmorrhage (Altwater, 1911 ; Greenacre, 1916), and cerebral injury (Duret, 1919). There are different theories to explain this, none of which is as yet sufficiently substantiated. Duret thought that a sudden displacement of cerebrospinal fluid by increased intracranial pressure compresses the vicinity of the aqueduct. Kolisko

(1911) pointed out that the basilar artery as it lies parallel to a bony surface is more exposed to the mechanical effect of increased pressure than other basal arteries. Greenacre (1916) put forward the theory that there must be some mechanism regulating the flow of blood to the brain through the carotid and vertebral systems respectively. As the result of massive ventricular hæmorrhage the mechanism becomes disturbed, and as a result a backflow of blood within the circle of Willis may take place. Wilson and Winkelmann (1926), who examined cases of pontine hæmorrhage occurring in different kinds of cerebral disease, pointed out the mechanical way in which the pons was exposed to damage by increased intracranial pressure. As our knowledge of the physiology of cerebral circulation is still incomplete, the problem of local vulnerability must await advances in physiology.

Conclusions

In a review of the pathology of cerebral apoplexy it is not possible to summarize the many facts which have been found or the theories which have been put forward to explain these facts. Surveying the development of the research on the subject of apoplexies the impression is obtained that a limit has been reached which purely descriptive investigation cannot pass. At least the morphology of the cerebral lesions found in apoplexy seems complete. It has been shown that the original mechanical conception of the early authors had to be abandoned in view of more recent findings, e.g. it now appears that cerebral softenings are not necessarily the result of physical obstruction of the arteries, and the theory that cerebral hæmorrhage is due to rupture of rigid or deformed vessels by internal strain is not sufficiently supported by facts. But such statements are essentially negative. The existence of a large number of different theories merely proves that none of them is adequate to explain all the facts. When the knowledge of the physiology of intracranial circulation has proceeded further a better explanation of events may be available. Nevertheless there are some points which may still be solved by a study of morphology. The rôle of the veins in the pathogenesis of apoplexy has been much neglected, due in part to the fact that little is known about the normal anatomy of the intracerebral veins. It is to be noted that the work of Pfeiffer has shown that the arrangement of the veins is essentially different in various parts of the cerebrum. This arrangement and the presence of many precapillary anastomoses in the brain make it necessary to re-investigate the subject from the angle of a study of the angioarchitecture of the brain.

An important point worthy of study is to determine if there is a close relationship between the character and the site of the lesion on one hand and the ætiology on the other. Already some work in this direction has been done by recent investigators. It appears probable that investigations directed to correlate the different clinical forms of hypertension and the pathology of the brain may prove fruitful. Further, as abnormalities in embryological development of the cerebral vessels appear to offer an explanation for the predilection of certain sites for hæmorrhage, a more intimate embryological study may be undertaken with advantage.

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OBITUARY NOTICE

S. A. KINNIER WILSON

THE name of Kinnier Wilson is to be numbered with those of the masters of the past. His death in his sixtieth year when, with enthusiasm and energy unabated, he was still at the height of his powers and engrossed in the completion of his *magnum opus*, is indeed a calamity to neurological science.

Wilson was essentially a clinician. From the day he entered the hospital wards as an undergraduate he showed a clinical aptitude which was remarkable. The manifestations of disease, their meaning and significance, entranced him. A quite exceptional observer, he literally devoured, in those early days, all literature available to him which had a bearing upon his personal experience. After an apprenticeship of six months as house physician to Byrom Bramwell in the Royal Infirmary, Edinburgh, he proceeded to Paris to pursue the speciality to which he proposed to devote his life. Gifted with a flair for languages and an exceptional appreciation of the niceties of expression, he took advantage to the full of his period of study with Pierre Marie and acquired an insight into and a sympathy with the brilliance of the French school of thought which coloured his future. When later he was attached as Resident Medical Officer, Assistant Physician, and Physician to the National Hospital in London, with which his life work and reputation were so intimately associated, he played a prominent part in maintaining the great traditions of that institution.

As an original observer and thinker, Wilson was pre-eminent. Profoundly influenced by his contacts with, and the writings of, Dr. Hughlings Jackson, for whom he had an admiration amounting almost to reverence, his trend of thought was reminiscent of that of the great English master. Wilson was not content with the elucidation of individual problems, for his interests and activities covered the whole field of neurology. If he excelled in his studies of the organic disorders, his contributions to the functional disorders were none the less illuminating. He will be remembered for all time in connection with his splendid work on progressive lenticular degeneration, the disease which he isolated and which bears his name. But many of his other contributions—as, for instance, his several writings upon the basal ganglia, aphasia, apraxia, and a score of other subjects—will live and would in themselves have made him famous as one of the leading neurologists of his day.

As a clinical teacher, too, Wilson was outstanding. Although he was apt at times to show a little not unnatural impatience with a particularly loquacious or unintelligent patient, his keen powers of observation, remarkable clinical memory, and singularly happy gifts of description and apt quotation fascinated his audience.

When after the war it was decided to institute a new journal in succession to the Review of Neurology and Psychiatry, founded by his father-in-law, Alexander Bruce, and continued by his brother-in-law, Ninian Bruce, it was singularly appropriate that Wilson should have been invited to act as Editor by the committee formed to discuss the continuance of a second British Journal of Neurology. He continued to act as such to the time of his death. During the last ten years the Journal was published under the ægis of the British Medical Association.

The Journal of Neurology and Psychopathology, the last number of which appeared a few months ago, had a character and complexion of its own determined by Wilson's outlook.

A striking figure which compelled attention, a quick tongue and a ready repartee, Sam Wilson showed at times a certain intolerance, for he did not suffer fools gladly, and an egotism which, though usually quite justified, did not pander to popularity. These characteristics were, however, counterbalanced by a keen appreciation of humour and a ready wit, often directed against himself, and those who knew him well and understood him overlooked his superficial mannerisms, for they knew that deep below the surface there lay a warm heart which never forgot.

A great neurologist has passed away. We mourn his passing, but his work remains as an incentive to the coming generations.

E. B.

EPITOME OF CURRENT JOURNALS

ACTA PSYCHIATRICA ET NEUROLOGICA

Vol. 12. Part 1. 1937.

The Psychoanalytical Technique and Therapy in the Light of Experimental Biology. Trygve Braatöy. 1.
Contributions à la Patho-physiologie de la Migraine. (Contributions to the patho-physiology of migraine.) Viggo Christiansen. 45.
Surgical Treatment of Postencephalitic Involuntary Movements of the Tongue. Byrnjulf Ingebrigtsen. 55.
Eine Studie über den Harnsäuregehalt des Liquor cerebro-spinalis bei einigen psychotischen Zuständen. (A study of the uric acid content of the cerebro-spinal fluid in some psychotic states.) G. Ingvarson. 61.
Troubles sui generis de la Motilité (en partie conditionnés organiquement). (*Sui generis* disturbances of movement.) Gabriel Langfeldt. 73.
*Polymyositis Acute and Subchronic, with Round-cell Infiltration of the Muscles. Ph. Levison. 89.
A Partial Abdominal Reflex of Pathological Type. G. H. Monrad-Khron. 97.

Polymyositis.—Two cases are described in which there was a prolonged history of pain and tenderness in the muscles. In one there was diminution of range of movement resulting from the tightness of the muscles. Muscle taken at biopsy revealed under the microscope extensive inflammatory processes. In the second case there was a lymphocytic infiltration around the blood vessels and an increase in the number of sarcolemmal nuclei. (L. E. A.)

AMERICAN JOURNAL OF PSYCHIATRY

Vol. 93. No. 5. March 1937.

A Review of Physico-Chemical Studies in Epilepsy. Meyer Brown and Harry A. Paskind. 1,009.
Conduct Disorders of Intellectually Subnormal Children. Louis A. Lurie. 1,025.
Folie à Deux. S. Kenneth Pollack. 1,039.
A Study of Cases of Folie à Deux. M. M. Grover. 1,045.
*Apraxias and Other Neurological Sequelæ of Carbon Monoxide Asphyxia. Ira C. Nichols and Margaret Keller. 1,063.
Biochemistry of the Psychoneuroses—A Review. R. A. McFarland and H. Goldstein. 1,073.
A Psychiatric Technique for the Examination of Criminals. Lowell S. Selling. 1,097.
The Age-Incidence Principle of Investigation in Evaluating the Biological Significance of Inherited Variations in the Problems of Human Constitution. William Washington Graves. 1,109.
*A Note of the Body Build of the Male Homosexual. Joseph Wortis. 1,121.
The Life and Work of Richard Maurice Bucke. George H. Stevenson. 1,127.
Group Activities on a Children's Ward as Methods of Psychotherapy. Lauretta Bender. 1,151.
Endocrine Disturbances in Behaviour Problems. Matthew Molitch. 1,175.
Studies in Endocrine Therapy in Epilepsy. Calvert Stein. 1,181.
Psychiatric Facilities in and about Pittsburgh. Cornelius C. Wholey. 1,185.
Psychiatric Studies in Medical Education. II. Neurotic Trends in Senior Medical Students. Edward A. Strecker, Kenneth E. Appel, Harold D. Palmer, and Francis J. Braceland. 1,197.
*Mortality among Patients with Involution Melancholia. Benjamin Malzberg. 1,231.

Apraxia in Carbon Monoxide Poisoning.—After a very brief summary of the literature, the authors report a case of recovery from carbon-monoxide poisoning. Most noticeable was the patient's inability to perform certain skilled acts. Agraphia was present. He was unable to learn easily, owing to severe disturbance of his visual motor centres. By verbalizing speech, it was possible to teach him to write. (H. C. I.)

Body Build of the Male Homosexual.—The author criticizes the earlier observations of Weil, who concluded that the greater height, longer legs, and wider hips of homosexuals were signs of constitutional eunuchoidism and femininity. When Weil's statistical survey of the configuration of homosexuals is compared with that obtained of normals by several observers, he finds that there is no actual evidence for any constant or typical intersexual traits among male homosexuals. (H. C. I.)

Mortality in Involution Melancholia.—An enquiry has been carried out of the mortality rate in involutional melancholia in the New York civil state hospitals. It was found that the annual crude death rate was 132·4 per 1,000 exposures. Diseases of the heart were the most common causes of death, being about eight times that occurring in the general population. (H. C. I.)

ARCHIV FÜR PSYCHIATRIE UND NERVENKRANKHEITEN

Vol. 106. No. 4. April 1937.

Der Wert der einfachen Kraniographie für die Erkennung endokranieller Drucksteigerungen. (The value of X-rays in the diagnosis of increased intracranial pressure.) W. Loepp. 410.
Irreleitende Symptomatologie bei einem Stirnhirngliom. (Misleading symptoms in a frontal lobe glioma.) H. Ganner. 436.
*Über die Histamin-empfindlichkeit der Paralytiker. (On the sensitivity to histamine in general paralysis.) Paul Buchler and Elemer von Sarudy. 461.
Über die nervösen und psychischen Störungen bei Pellagra. (Nervous and mental disturbances in pellagra.) C. Mayer. 472.
Ein Fall von Pellagra in Bayern. (A case of pellagra in Bavaria.) H. Ganner. 495.

Sensitivity to Histamine in General Paralysis.—Patients affected by G.P.I. are particularly sensitive to the application of histamine, in contrast to other groups of neurological and mental disorders. This increased sensitivity is regarded as constitutional and it may be one of the factors which predispose to the development of the disease. (A. M.)

Vol. 106. No. 5. June 1937.

Über die Bedeutung der prämorbiden Persönlichkeit für Verlauf und Gestaltung der Psychose. Die zwangsneurotische Persönlichkeit im schizophrenen Prozess. (On the significance of the præmorbid personality in the structure of psychosis. The obsessional personality in schizophrenia.) Erwin Stengel. 509.
Zur Reaktionslage des reticuloendothelialen Systems (RES) von Geisteskranken insbesondere Schizophrenen. (The reaction of the reticulo-endothelial system in the psychoses, especially schizophrenia.) V. Nicolajev, Fr. Tebelis, and V. Osolin. 554.
*Angiographischer Befund bei Pickscher Atrophie. (Angiographic findings in Pick's disease.) Benedek, Ladislaus, and Bela Horányi-Hechst. 563.
*Über das Elektrenkephalogramm des Menschen. (The electro-encephalogram in man.) Hans Berger. 577.
Über Verlauf und Prognose der Schizophrenie. (Katatonie, Hebephrenie, Dementia simplex.) (On the course and prognosis of schizophrenia, catatonia, hebephrenia, and dementia simplex.) Walter Gerloff. 585.

The Præmorbid Personality in the Structure of Psychosis.—Schizophrenic psychoses superseding præmorbid obsessional phenomena take a rather benign course. The author concludes from this that obsessional mechanisms exert an inhibitory and "immunizing" influence upon the psychosis. The removal of obsessional symptoms should, therefore, not be attempted in the treatment of the psychosis. (A. M.)

Angiographic Findings in Pick's Disease.—A patient aged 50, with the clinical symptoms suggestive of frontal lobe lesion, is submitted to encephalography, cerebral puncture, and angiography. The encephalogram shows greatly enlarged subarachnoid spaces in both the frontal regions. Tissue obtained by puncture is histologically examined. The ganglion cells show vacuolic degeneration but no Alzheimer's fibril change and no argentophile inclusions. A few senile plaques are found. In the cerebral arteriogram striking changes are seen in the regions supplied by the anterior cerebral artery, but none in that of the middle cerebral artery. (A. M.)

The Electro-encephalogram in Man.—The author supplies evidence obtained from pharmacological experiments with caffeine, cocaine, atropine, that the so-called beta-waves are to be regarded as a material substrate accompanying psychological processes. (A. M.)

ARCHIVES OF NEUROLOGY AND PSYCHIATRY

Vol. 37. No. 4. April, 1937.

The Psychobiologic Unit as a Pattern of Community Function. George S. Stevenson. 742.
Amnesia. R. D. Gillespie. 748.
Psychiatry in China. R. S. Lyman. 765.
Latency of Cortical and Retinal Action Potentials Induced by Illumination of the Eye. Ging-Hsi Wang. 772.
*Cerebral Circulation : XLV. Vasodilation in the Pia Following Stimulation of the Geniculate Ganglion. Henry S. Forbes, Gladys I. Nason, Stanley Cobb, and Ruth C. Wortman. 776.
Psychic Phenomena in Association with Cardiac Failure. Nolan D. C. Lewis. 782.
The Psychopathology of Metaphor. Wendell Munciel 796.
*Aversion and Negativism. Oskar Diethelm. 805.
Relationship of Declining Intelligence Quotients to Maladjustments of School Children. Esther Loring Richards. 817.
The Pathogenesis of Hallucinations and Delusions : Remarks on the Distinction between Pathogenesis and Etiology in Psychiatry. Max Levin. 839.
Contribution to the Physiology of the Conditioned Reflex. W. Horsley Gantt. 848.
The Dilemma of Growth. Frederick H. Allen. 859.
The Type of Neurotic Hypomanic Reaction. Bertram D. Lewin. 868.
Modifications in a Schizophrenic Reaction with Psychoanalytic Treatment. Lawrence S. Kubie. 874.
Studies in Schizophrenia : Chemical Analyses of Blood and Cerebrospinal Fluid. S. Katzenelbogen. 881.
*Personality Features and Reactions of Subjects with Migraine. Harold G. Wolff. 895.
Prognosis in Child Psychiatry. Leon Kanner. 922.
Syphilitic Arachnoiditis of the Optic Chiasm. Louis Hausman. 929.
*Colloid Cyst of the Third Ventricle : Report of a Case : Operative Removal with Section of Posterior Half of Corpus Callosum. John H. Trescher and Frank R. Ford. 959.
The Catathymic Crisis: A Clinical Entity. Frederick Wertham. 974.

Cerebral Circulation.—Continuing their studies of the cerebral circulation in cats, the authors found that stimulation of the facial nerve at the geniculate ganglion causes dilatation of arteries in the pia covering the parietal region. This dilatation contrasts sharply with that which

results from a fall of blood pressure; the vasodilation associated with stimulation of the facial nerve occurs when the blood pressure is normal at the outset and remains constant throughout the period of stimulation; the response is strictly ipsilateral and is abolished by cocaine.

The vasodilation associated with fall of blood pressure occurs only when the latter reaches a critical level; it involves the pial arteries of both hemispheres and is not affected by cocaine.

It seems probable that higher mammals (cats and monkeys) possess a true vasodilator innervation of the arteries supplying the parietal cerebral cortex. The nerve fibres involved come from the medulla and reach their destination via the facial nerve, the great superficial petrosal nerve and, in monkeys at least, the internal carotid nerves. (R. M. S.)

Aversion and Negativism. — In contrast to negativistic reactions, with their automaticity and absence of sufficiently intelligible motivation, aversion reactions are characterised by emotions and motives which make them intelligible to the patients and to others. The outstanding emotions are resentment, dislike, hate and disgust. Aversion is the reaction of an unbending personality to an unacceptable and unconquerable situation. While negativism is the expression of autism and submission, aversion seems to be related to aggression and self assertion. The psychopathologic picture varies individually, the most frequent aversion reactions being sullen or resentful unco-operativeness, stuporous and paranoid conditions and non-recoverable rut formations in association with depressions in the involutional and later periods of life. By studying the factors which cause aversion the physician will frequently find ways to make the situation more tolerable to the patient and modify an unbending attitude by treatment. (R.M.S.)

Personality Features and Reactions of Subjects with Migraine.—A psychobiologic study of 46 subjects with migraine has revealed that these persons were so constituted as to be peculiarly prone to the development of pernicious emotional states, either sustained or with acute episodic exacerbations. In certain of them a sudden increase in stress was provoked by clearly definable life situations. Such periods were associated with an increase in the intensity and frequency of attacks of migraine. (R. M. S.)

Colloid Cyst of the Third Ventricle.—A colloid cyst of the third ventricle was removed by operation together with the posterior half or more of the corpus callosum. Reasons are given in support of the belief that section of the corpus callosum was the only significant injury inflicted on the brain. From an analysis of the symptoms after the operation, the writers conclude that a lesion of the posterior part of the corpus callosum causes alexia on the left and tactile agnosia for letters in the left hand, just as a lesion of the anterior part of the corpus callosum causes motor apraxia of the left arm. It is suggested that the symptoms described constitute the syndrome of the posterior part of the corpus callosum. (R. M. S.)

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*Athetosis. II. Surgical Treatment of Unilateral Athetosis. Paul C. Bucy and Theodore J. Case. 983.
Anatomic and Pneumographic Studies of the Temporal Horn, with a Further Note on Pneumographic Analysis of the Cerebral Ventricles. A. E. Childe and Wilder Penfield. 1,021.
*Serum Disease of the Nervous System : Report of Three Cases. Walter M. Kraus and L. Beverley Chaney. 1,035.
Nature and Significance of Multiple Petechial Hemorrhages Associated with Trauma of the Brain. Walter F. Schaller, K. Tamaki, and Henry Newman. 1,048.
Contribution made by Roentgenographic Evidence after the Injection of Iodized Oil. Joseph H. Globus. 1,077.
*Neuroptic Myelitis versus Multiple Sclerosis : A Pathologic Study. George B. Hassin. 1,083.
*Primary Demyelinating Processes of the Central Nervous System : An Attempt at Unification and Classification. Armando Ferraro. 1,100.
Chronic Bilateral Subdural Hematoma : Encephalographic Diagnosis with Report of Three Cases. William L. Holt, Jr., and Grosvenor B. Pearson. 1,161.

Surgical Treatment of Unilateral Athetosis.—Two cases of athetosis are presented; one of much more severe athetosis on one side, the other of strict hemiathetosis. In both instances a large dose of barbiturates temporarily abolished the involuntary movements, leaving the voluntary movements more or less intact. In both instances extirpation of the " arm region of area 6 " (Brodmann) resulted at first in complete abolition of the involuntary movements, which later returned to a mild degree. In case 2 this was attributed to the sparing of part of the representation of the fingers in area 6; in case 3, to the careful sparing of all of area 4. From these observations it is concluded that: (1) the involuntary movements of athetosis are projected largely from area 6; (2) the parapyramidal fibres from area 4 may participate to some slight extent in the production of these movements; and (3) in suitable cases of hemiathetosis, or of athetosis predominating on one side of the body, extirpation of the contralateral area 6 or areas 4 and 6 is an appropriate therapeutic procedure which gives gratifying results.

In the discussion which followed the presentation of this paper, it was pointed out that the involuntary movements exhibited by case 2 were not typical of athetosis. (R. M. S.)

Serum Disease of the Nervous System.—The ill effects on the nervous system following the use of foreign sera for therapeutic purposes are discussed under the headings cerebral, spinal, radicular, and neural syndromes. The most frequent, serious, and long-lasting syndrome is that of paralysis of the muscles supplied by the fifth and sixth cervical spinal roots. The underlying pathological process is considered to be a primary involvement of the blood vessels, such as occurs in the ordinary type of serum disease. (R. M. S.)

Neuroptic Myelitis.—Hassin reports the case of a woman aged 33 years who developed blindness, paralysis of the lower extremities, severe bladder and sensory disturbances, and extensive decubitus. The illness, which lasted fourteen months, was marked by a short remission. The pathological changes in the nervous system consisted of a combined patchy degeneration of the spinal cord and the visual fibres. Macroscopically, the patches resembled those of multiple sclerosis, but differed microscopically in the scarcity or absence of glia fibres, the simultaneous involvement of the grey or white substances, and the massive cellularity. Neuroptic myelitis, of which this was an example, is a definite toxic infectious disease process, differing both from multiple sclerosis and disseminated encephalomyelitis. (R. M. S.)

Primary Demyelinating Processes.—In a review of 52 pages, Ferraro discusses the various types of primary demyelinating processes seen in the central nervous system and attempts a classification which is based on both the clinical and the pathological characteristics of the various conditions analysed. The paper, which does not lend itself to abstraction, constitutes a very valuable résumé of a number of diseases which are probably fundamentally identical in their histopathology. (R. M. S.)

Vol. 37. No. 6. June 1937.

*Role of the Cerebellum in Postural Contractions. H. W. Magoun, W. K. Hare, and S. W. Ranson. 1,237.
*Agenesis of the Corpus Callosum : Its Recognition by Ventriculography. Olan R. Hyndman and Wilder Penfield. 1,251.
Reduction of Postencephalographic Symptoms by Inhalation of 95 per cent. Oxygen. Robert S. Schwab, Jacob Fine, and William Jason Mixter. 1,271.
*Anomalous Commissure of the Third Ventricle (Aberrant Dorsal Supra-Optic Decussation) : A Report of Eight Cases. A. R. Vonderahe. 1,283.
*Autonomic Innervation of the Eyelids and the Marcus Gunn Phenomenon : An Experimental Study. F. H. Lewy, Robert A. Groff, and Francis C. Grant. 1,289.
*Evidences of Vascular Occlusion in Multiple Sclerosis and "Encephalomyelitis." Tracy J. Putnam. 1,298.
Health as a Psychic Experience. Paul Schilder. 1,322.
*Structure of the Nerve Root. II. Differentiation of Sensory from Motor Roots ; Observations on Identification of Function in Roots of Mixed Cranial Nerves. I. M. Tarlov. 1,338.
Ganglioglioneuroma of the Spinal Cord Associated with Pseudosyringomyelia : A Histologic Study. Ben W. Lichtenstein and Howard Zeitlin. 1,356.
Marie's Ataxia (Olivopontocerebellar Atrophy) : Clinical and Pathologic Considerations. George B. Hassin. 1,371.
Vibration Sense. Lewis J. Pollock. 1,383.

Cerebellum in Postural Contractions.—The prolonged maintenance of rebound excitation following cerebellar stimulation in the normal cat is considerably reduced by deafferentation of the reacting limb and appears to be decreased a little more by labyrinthectomy in addition to deafferentation. After the elimination, however, of all the afferent impulses on which the tonic effects of the stretch and labyrinthine reflexes depend, continued discharge of both flexor and extensor centres for the fore limb has been observed for periods of up to a minute or longer. Release effects have been produced by destruction of the cerebellum in several ways and have been compared with the rebound contractions which follow the cessation of cerebellar stimulation. In a number of respects the two do not appear to be homologous. (R. M. S.)

Agenesis of the Corpus Callosum.—For the first time, living patients presenting agenesis of the corpus callosum are reported, together with a description of the ventriculogram. Of the five, two presented complete and two partial absence of the commissure ; one presented absence of only the splenium of the corpus callosum.

Analysis of the structures and the embryologic principles involved makes it possible to identify the lines of the ventriculographic shadows and to distinguish complete from incomplete agenesis. The chief characteristics of the ventriculograms in the anteroposterior view are symmetrical separation of the anterior horns, with a moniliform shadow of gas between them which is continuous with the third ventricle below and rises higher than this ventricle should. Most striking of all is an almost right-angled shelving or bicornuate appearance of the bodies of the lateral ventricles. In the opinion of the authors, the latter is pathognomonic of the condition. (R. M. S.)

Anomalous Commissure of the Third Ventricle.—Vonderahe describes eight cases of an anomalous commissure of the third ventricle, intimately related to the paraventricular nucleus. The cells of the latter are found in it in greater or less number and the fibres which pass through it appear to be associated with the dorsal supra-optic decussation (Ganser's commissure). A study of the anomaly suggests that some of the fibres of the dorsal supra-optic decussation enter the paraventricular nucleus of the opposite side, thus pointing to a probable termination of these fibres in normal brains. (R. M. S.)

Gunn Phenomenon.—The Marcus Gunn phenomenon is characterized by ptosis, usually congenital, of the affected eye by slow elevation of the upper eyelid synchronous with chewing movements.

In a case investigated by one of the writers the condition was completely relieved by section of the motor root of the fifth nerve, a result which prompted the experimental investigations now reported.

From 7 to 42 days after intracranial section of the third nerve, it was found that intra-arterial injection of acetylcholine caused widening of the palpebral fissure on the side of operation. The same reaction, to a less marked degree, was produced by intra-arterial injections of nicotine, but epinephrine produced enlargement of both palpebral fissures with dilatation of the pupils and general systemic effects. Faradic stimulation of the peripheral stump of the first division of the fifth nerve, but not of the second or the third division, caused a reaction exactly similar to the injection of acetylcholine into the ipsilateral carotid artery. Epinephrine inhibited this reaction in some cats, while in others no effect was noted. Atropine did not inhibit the reaction, regardless of the size of the dose.

The conclusion is reached that centrifugal autonomic fibres in the peripheral divisions of the fifth nerve are concerned in the production of this pseudomotor eyelid phenomenon, but at present it cannot be determined whether these fibres are sympathetic or parasympathetic. (R. M. S.)

Vascular Occlusion in Multiple Sclerosis.—The author produces histological evidence in support of the view that the lesions of multiple sclerosis and of the forms of disseminated encephalomyelitis which seem to represent a more acute stage of the process in the former disease, are produced by a local circulatory disturbance, apparently of the nature of an obstruction on the venous side. According to this point of view, the primary abnormality is probably in the clotting mechanism of the blood. (R. M. S.)

Structure of the Nerve Root.—Tarlov believes that a study of the differences in structure of the various cerebrospinal nerve roots affords criteria for differentiating sensory and motor nerve roots.

Thus, motor nerves differ grossly from sensory nerves in being composed of a greater number of fine filaments at their superficial origin. Microscopically, the sensory roots contain longer glial segments than the motor roots, the latter usually being less cellular and exhibiting to a less extent the phenomenon of cell piling at the transition zone.

On the basis of these criteria, the nervus intermedius of Wrisberg is a mixed nerve, usually containing separate motor and sensory roots. The involuntary motor roots are distinct from the main voluntary motor portion of the facial nerve.

The glossopharyngeal nerve usually contains separate motor and sensory roots.

The vagus-spinal accessory complex often consists of two distinct motor portions, the one contained in the small ventromedial roots and the other in the large caudal roots. The cephalic portion of this complex is chiefly sensory.

Varying degrees of approximation and even fusion of the rootlets of the nervus intermedius, glossopharyngeal nerve, and vagus-spinal accessory complex may occur. (R. M. S.)

BRAIN

Vol. 60. Part 2. April 1937.

Chrondro-osteo-dystrophy of the Hurler Type (Gargoylism). A Pathological Study. W. R. Ashby, R. M. Stewart, and J. H. Watkin. 149.
Toxic Hydrocephalus. Douglas McAlpine. 180.
*The Axis-cylinder as a Pathway for Dyes and Salts in Solution, with Observations on the Node of Ranvier in the Rabbit. J. R. Perdrau. 204.
*The Origin of the Raised Pressure of the Cerebrospinal Fluid which accompanies Sub-tentorial Tumours. T. H. B. Bedford. 211.
*Observations on the Roentgen Treatment of Intracranial Gliomata, with Especial Reference to the Effects of Irradiation upon the Surrounding Brain. John E. A. O'Connell and Alexander Brunschwig. 230.
A Clinical Study of the Respiratory Movements in Hemiplegia. Lawrence C. Kolb and François Kleyntjena. 259.

The Axis-cylinder as a Pathway for Dyes and Salts.—The author reports the results of experiments undertaken to show if the axis-cylinder was in fact a possible pathway for neurotropic viruses. Using mainly the sciatic nerve of the rabbit, he immersed one end of the nerve in a solution of potassium ferrocyanide and ammonium citrate. Diffusion of the substance up the axis-cylinder was subsequently demonstrated by fixing the tissue in formalin and dilute HCl. Permeation was increased by cataphoresis and occurred both in the living and the excised nerve. Colloidal substances did not permeate the nerve. The method also demonstrated that the axis-cylinder is composed of material like soft jelly which is denser and thinner at the nodes of Ranvier. (J. D.)

Raised Pressure of the Cerebrospinal Fluid with Sub-tentorial Tumours.—The author produced extra-dural sub-tentorial tumours in dogs by packing waxed cotton-wool through a small trephine hole. On the sixth day the cisternal pressure and in some cases the ventricular pressure were measured and then the animals were sacrificed. An increased cerebrospinal

fluid pressure was found in 19 out of 21 cases. The possible causes of this increase of pressure are reviewed and excluded as far as possible. Post-mortem revealed no inflammatory reaction around the tumour and no evidence was found that the cerebral cortex was compressed against the absorbing surface of the arachnoid. The venous pressure in the torcula was normal and the vein of Galen was intact. Simultaneous intraventricular and cisternal pressure readings showed a free communication between those two points, while injections of dye into the cistern showed a normal spread upwards over the cortex. The author concludes that the cause of the increased cisternal pressure was either overproduction of cerebrospinal fluid or that an intermittent block between the cistern and the base of the brain existed. (J. D.)

Roentgen Treatment of Intracranial Gliomata.—The authors review the literature on this subject and describe the effects of irradiation on normal brain tissue and on tumour tissue. The former showed changes not observed in other circumstances. These consisted of a swelling of the nerve cells, which contained a great excess of lipochrome pigment, and hypertrophy of the astrocytes. The nerve fibres were normal and there was but little change in the vessels. Further evidence that Roentgen therapy injures normal brain tissue was found in the clinical history of certain cases which became cachetic and died following treatment. By comparing biopsy specimens taken before treatment with those secured at post-mortem they found that irradiation has no effect on relatively benign tumours such as fibrillary astrocytomata and ependymomata. It was impossible to be certain of the reaction of glioblastoma multiforme, but clinical experience taught that irradiation had a beneficial effect. Medulloblastomata gave the most satisfactory response both from the pathological and the clinical point of view. They suggest the use of Roentgen therapy post-operatively to control anaplastic degeneration in the more benign growths and emphasize the importance of an accurate pathological diagnosis before treatment is commenced. Their present practice is to deliver a tumour dose of 4,000 to 4,500 r. by one or more portals and they point out the disadvantages of using a single large dose. (J. D.)

BULLETIN OF THE NEUROLOGICAL INSTITUTE OF NEW YORK

Vol. 6 No. 2. August 1937.

Some Stages in the Development of the Neural Complex in *Ecteinascidia turbinata*. Adolph Elwyn. 163.
*Insulin Response in Acromegaly. Benjamin N. Berg. 178.
*Pituitary Basophilism of Cushing. Syndrome of the Basophilic Adenoma. Irving Pardec. 183.
*Basal Metabolism in Organic Psychoses. J. Notkin. 199.
Cerebral Sequelæ of Severe Jaundice in the Newborn. Walter O. Klingman and Earl R. Carlson. 228.
*The Sense of Vision : Introduction. Charles A. Elsberg. 233.
I. A Method for the Study of Acuity of Vision and of Relative Visual Fatigue. Charles A. Elsberg and H. Spotnitz. 234.
II. The Reciprocal Relation of Area and Sight Intensity and its Significance for the Localization of Tumours. Charles A. Elsberg and H. Spotnitz. 243.
III. A Theory of the Functions of the Retina. Charles A. Elsberg and H. Spotnitz. 253.
*A Study of Thresholds in Apperception of Passive Movement among Normal Control Subjects. Robert W. Laidlaw and Mary Alice Hamilton. 268.
*Extramedullary Tumours of the Upper Cervical Portion of the Spinal Cord. Samuel E. Soltz and George A. Jervis. 274.
Calcified Subpial Lesions of the Spinal Cord with Associated Varicose Veins. Clarence C. Hare and William H. Everts. 295.
Meningioma : Report of an Unusual Case. Leo M. Davidoff. 300.
*Granulomatous Encephalomyelitis due to an Encephalitozoon (Encephalitozoic Encephalomyelitis). A New Protozoan Disease of Man. Abner Wolf and David Cowan. 306.
Manual Reflex : The Ulnar Adductor Reflex. J. Lawrence Rool. 372.
The Symptom of Electric Shock in Multiple Sclerosis. Leon A. Salmon. 378.
A Note on the Effect of Heat and of Cold upon Certain Symptoms of Multiple Sclerosis. Donald J. Simons. 385.

Insulin Responses in Acromegaly.—Six subjects with acromegaly were studied to determine if the blood-sugar response to insulin varied from normal. 0·067 units of insulin per kilogram of body weight were given. Larger doses were also given. Blood samples were obtained before, 20, 30, 40, 60, 90, and occasionally 120 minutes after. In one group of these subjects the blood-sugar changes were within the limits obtained in normal controls. In the remaining a marked resistance to insulin was found. It is apparent that the "anti-insulin factor" of the gland is not similarly involved in all the cases. (L. E. A.)

Basophilism of Cushing.—The author brings together all the literature related to this subject. This is an excellent and full bibliography. (L. E. A.)

Metabolism in Organic Psychoses.—This is a long article dealing with basal metabolic rate readings in 94 subjects with arteriosclerotic psychosis, 36 with senile psychosis, 65 with untreated dementia paralytica, 106 with treated dementia paralytica, 48 with alcoholic psychosis, and a small group with various types of psychosis. In the first two groups the majority of subjects had a basal metabolic rate on the minus side. There was a tendency to have a higher rate in the treated cases of dementia paralytica. In the alcoholic group the majority of readings were on the

minus side. After discussing the findings, the author comes to the conclusion that "the lower oxygen consumption must be considered as secondary to the cellular alterations." (L. E. A.)

Sense of Vision.—The authors have approached the subject anew and have devised tests to determine the effects of unilateral and bilateral stimulation of the retina by light upon acuity of monocular and binocular vision. They describe in detail their apparatus (illustrated). After discussing some observations, their final paper deals with a theory of the functions of the retina. These papers do not lend themselves to abstraction. (L. E. A.)

Threshold for Passive Movement.—With an apparatus to determine perception of movement a study of a series of normal subjects has been undertaken. They found a wide range of variation in normal, but this variation became more marked in a group of older people. They have worked out threshold values for several joints which will serve as an excellent starting-point to a study of subjects with disease of the nervous system. (L. E. A.)

Tumours of Upper Cervical Cord.—Five cases of extramedullary tumour situated in the high cervical region are presented and the symptomatology analysed. The authors lay special emphasis on the unusual sensory signs. They varied in each case; in two, numbness in the upper limbs was present at the onset. Wasting was limited commonly to the muscles of the shoulder girdle, while weakness of the diaphragm occurred in two cases. They discuss in detail the differential diagnosis. (L. E. A.)

Granulomatous Encephalomyelitis.—This case is of special interest because of the extreme rarity of the condition and the resemblance of the parasitic agent to one found in rabbits. It was in an infant 24 days old admitted because of convulsions. The child was running a temperature and died 6 days later. The lumbar and ventricular fluid contained a large number of cells interpreted as lymphocytes in the lumbar fluid, as disintegrating cells or polymorphs in the ventricular fluid. Two eosinophil cells were seen. On post-mortem examination the changes in the nervous system were of particular interest. These consisted of granulomas, massive areas of infiltration, and necrosis, chiefly in relation to the ventricular walls. Similar lesions were found in the choroid and retina. An organism was also found which bore a striking resemblance to the *Encephalitozöon cuniculi*. (L. E. A.)

CLINICAL SCIENCE

Vol. 3. No. 1. July 1937.

Investigations of the Functions of the Small Intestine in Man by Intestinal Intubation. W. H. Owles.
Part I. The Technique of Intestinal Intubation in Man. 1.
Part II. Determination of Diastase, Invertase, Erepsin, Lipase and Lactose in the Pure Juice of the Small Intestine. 11.
Part III. Factors Influencing the Secretion of Juice by the Small Intestine. 21.
The Effect of Large Doses of Iron in the Absorption of Phosphorus. John Fleming Brock. 37.
*Observations on a Case of Familial Periodic Paralysis. R. S. Aitken, E. M. Allott, L. I. M. Castleden, and Mary Walker. 47.
*The Effect of Asphyxia and Cocaine on Nerves belonging to the Nocifensor System. Thomas Lewis. 59.
*The Double Pain Response of the Human Skin to a Single Stimulus. Thomas Lewis and E. E. Pochin. 67.
*The Action of Choline Esters in Myasthenia Gravis. F. R. Fraser, Murray McGeorge, and G. E. Murphy. 77.

Observations on a Case of Familial Periodic Paralysis.—The original observation by Walker and Allott that during the paralytic stage the serum potassium value was low has been confirmed and further investigations carried out. By a further study of the same subject the authors have found that the serum potassium may be lowered abnormally by the administration of glucose by mouth, by the injection of insulin, and by a combination of the two. If the serum potassium falls below 12 mgm. per 100 c.c. paralysis sets in and the paralysis may be relieved by the administration of potassium chloride by mouth. Following the administration of potassium, the blood potassium level rises. (L. E. A.)

Effect of Asphyxia and Cocaine on Nerves belonging to the Nocifensor System.—Further observations have been made on the state of hyperalgesia occurring as the result of a crush of the skin. The author states in his summary: "The development of hyperalgesia around a small crush of the skin can be prevented by asphyxiating the skin; and the spread of hyperalgesia can be blocked by asphyxiating a narrow band of skin. The duration of asphyxia required is such as has little appreciable effect on the functions of the sensory nerves of the skin. Asphyxia must be carried a little further to prevent the development of the 'flare' or to interfere with the spread of the 'flare' in response to the same injury. Asphyxia stops hyperalgesia from developing by paralysing the nerves concerned: these nerves are paralysed before the sensory nerves. Cocaine introduced electro-phoretically into the skin paralyses the pain nerves completely and the touch nerves entirely or almost entirely before it interferes with the hyperalgesia reaction. The

conclusion previously found, that the nocifensor nerves, though belonging to the posterior root system, are not sensory but belong to a special system, is supported by both these observations." (L. E. A.)

Double Pain Response of the Human Skin to a Single Stimulus.—Further observations have been carried out on the double pain response previously investigated by Thalberg. By producing ischemia in the limbs and by anæsthetization with cocaine it has been possible to dissociate the two responses. These results are interpreted as indicating that the two pain responses are mediated by two sets of nerves, the one set being fast conducting and the other slow conducting. (L. E. A.)

Action of Choline Esters in Myasthenia Gravis.—In a study of two subjects suffering from myasthenia gravis and who had had very prolonged therapy with prostigmin. It was found that the subcutaneous administration of acetyl choline, acetyl B-methyl choline, and carbaminoyl choline caused some delayed recovery of muscle power. From their observations the authors suggest that there is a defect in the production of acetylcholine in myasthenia gravis. (L. E. A.)

JOURNAL BELGE DE NEUROLOGIE ET DE PSYCHIATRIE

Vol. 37. No. 4. April 1937.

Remarques Cliniques sur les Tumeurs du Lobe Temporal. (Clinical observations in temporal lobe tumours.) J. A. Barré. 209.
Les Troubles Cochléires dans les Tumeurs du Lobe Temporal. (Disturbances of hearing in temporal lobe tumours.) J. A. Barré. 229.
Les Troubles Vestibulaires et de L'Equilibre Statique dans les Tumeurs du Lobe Temporal. (Disturbances of equilibration in temporal lobe tumours.) J. A. Barré. 238.
Troubles de L'Odorat et du Goût comme Signe d'une Lésion Focale du Lobe Temporal. (Disturbances of smell and taste as a sign of a focal lesion of the temporal lobe.) R. Brun. 262.

Vol. 37. No. 5. May 1937.

Trois Cas de Tumeur de la Région Épiphysaire. (Three cases of tumour in the region of the pineal.) Paul van Gehuchten and Paul Callewaert. 283.
*Tumeurs Cérébrales Multiples et Troubles Mentaux. (Multiple cerebral tumours and mental symptoms.) M. Leroy. 292.
*Sur une Forme Particulière de Gliomatose Périvasculaire. (On a form of perivascular gliomatosis.) H. J. Scherer and J. de Busscher. 299.
Contribution à L'Étude du Syndrome Polyradiculonévritique de Guillain et Barré chez L'Enfant. (Contribution to the study of the polyneuritis of Guillain and Barré in an infant.) Joseph Ansay. 311.

Multiple Cerebral Tumours and Mental Symptoms.—The author draws attention to the prevalence of mental symptoms as an early sign of multiple cerebral tumours. The content of the mental symptoms vary considerably, but are of progressive character. (D. D.-B.)

Perivascular Gliomatosis.—The authors describe an unusual form of glioma in which the cells are collected around the blood vessels. They consider in detail the character of these cells in the light of the recent staining methods. There was also a marked vascular proliferation. (D. D.-B.)

Vol. 37. No. 6. June 1937.

*La Microglie Normale chez le Singe (*Macacus rhesus*). (Normal microglia in the monkey.) A. Dewulf. 341.
Troubles Mentaux Consécutifs à une Commotion Cérébrale avec Gliome. (Mental disturbances following trauma in a case with glioma.) J. Radermecker. 366.
*Action de la Prostigmine dans un Cas de Myasthénie à Forme de Dystrophie Musculaire Progressive. (The action of prostigmine in a case of myasthenia combined with a form of progressive muscular dystrophy.) L. Laruelle and L. Massion-Verniory. 376.
*Catalepsie Insulinique Réglable chez la Souris. (Catalepsy produced by insulin in mice.) P. Divrey and E. Evrard. 382.
A Propos des Recherches Généalogiques dans les Démences. (Some notes on genealogical studies in dements.) H. Hoven. 393.
Deux Cas de Myélite-cécité. (Two cases of syphilitic optic atrophy with myelitis.) B. Dujardin. 399.

Normal Microglia in the Monkey.—A prolonged and careful study of the microglial cells as shown up by the Hortega method has been undertaken. The article is well illustrated and requires to be read in detail. (D. D.-B.)

The Action of Prostigmine in Myasthenia.—The authors report improvement in a case of myasthenia in which there has been administration of prostigmine. They have no relevant observations dealing with the cause of the disease. (D. D.-B.)

Catalepsy in Mice.—By means of the injection of insulin, it is possible to bring about a state of inertia or " psycho-motor stupor " in mice. These results closely resemble those obtained with bulbocapnin. (D. D.-B.)

JOURNAL FÜR PSYCHOLOGIE UND NEUROLOGIE

Vol. 47. No. 6. June 1937.

Die myeloarchitektonische Gliederung des Stirnhirns beim Menschen und beim Schimpansen. I Teil: Myeloarchitektonische Gliederung des menschlichen Stirnhirns. (The myeloarchitecture of the frontal cortex in man and chimpanzee. I. Myeloarchitecture of the human frontal cortex.) Eduard H. Strasburger. 461.

*Quantitative Untersuchungen über den Kapillarengehalt der grauen Substanz des Gehirns bei jungen und bei alten Menschen. (Quantitative investigations of the capillaries of the grey matter of the brain of young and old human subjects.) Ake G. H. Lindgren. 492.

Die bioelektrische Tätigkeit der Grosshirnrinde im normalen Schaf und in der Narkose durch Schlafmittel. (The bioelectrical activity of the cerebral cortex during normal sleep and during general anæsthesia produced by hypnotics.) R. Klaue. 510.

Zur Unabhängigkeit der einzelnen Eigenschaften der Manifestierung einer schwachen polaren Genmutation (Venæ abnormes) bei *Drosphila melanogaster*. (The independence of the single qualities of the manifestation of a weak polar gene mutation (venæ abnormes) in *Drosophila melanogaster*.) Marguerite Vogt. 532.

Zur analyse der Musikalität: Rhythmusproblem. Erste Mitteilung. (Contribution to the analysis of musical faculty: the problem of rhythm.) Claus-Viktor Loebe. 550.

Capillaries of the Grey Matter.—Five brains of patients of 16 to 81 years were examined. Histological sections were made without injection of the vessels. The capillaries were demonstrated by staining the red blood cells. The length of the capillaries in identical areas was measured by a special method of counting and brought into a mathematical relation to the weight of the brain. The amount of capillaries is in direct proportion to the volume of the parenchyma. The decrease of capillaries is probably caused by capillary fibrosis. It is not certain whether the diminution of brain substance is the result or the cause of the diminution of the capillaries. (K. S.)

JOURNAL OF ANATOMY

Vol. 71. Part 3. April 1937.

*The Projection of the Medial Geniculate Body to the Cerebral Cortex in the Macaque Monkey. A. Earl Walker. 319.

An Experimental Investigation of the Cerebral Hemispheres of *Lacerta viridis*. F. Golby. 332.

The Narial Margins in Man. Edwin H. Johnson. 536.

On the Cheek-bones in Teleostome Fishes. T. S. Westoll. 362.

The Ossification of the Human Frontal Bone. Verne I. Inman and John B. de C. M. Sanders. 383.

The Interosseous Muscles of the Hand. C. R. Salisbury. 395.

The Sheath of the Rectus Abdominis. Robert Walmsley. 404.

Projection of the Medial Geniculate Body to the Cerebral Cortex.—*Author's Summary.*—From a study of the retrograde cell degeneration occurring in the medial geniculate body after lesions of the temporal cortex in the macaque monkey it is concluded: (1) That the projection of the medial geniculate body to the cerebral cortex is confined to a small area in the superior surface of the superior temporal gyrus. (2) That there are distinctive spatial relationships between the medial geniculate body and the cerebral cortex. (L. E. A.)

JOURNAL OF CLINICAL INVESTIGATION

Vol. 16. No. 3. May 1937.

Variations in the Permeability of the Red Blood Cells in Man, with Particular Reference to the Conditions obtaining in Pernicious Anæmia. Ole Bang and Søren L. Ørskov. 279.

The Influence of Muscular Exercise on Blood Sugar Concentrations. Florence H. Smith and K. A. Smith. 289.

Clinical Studies of the Blood Volume. (1) Clinical Application of a Method employing the Blue Azo Dye "Evans Blue" and the Spectrophotometer. John G. Gibson and William A. Evans. 301.

Clinical Studies of the Blood Volume. (2) The Relation of Plasma and Total Blood Volume to Venous Pressure, Blood Velocity Rate, Physical Measurements, Age, and Sex in 90 Normal Humans. John G. Gibson and William A. Evans. 317.

Relation of Thyroid and Parathyroid Glands to Calcium and Phosphorus Metabolism. Study of a Case with Coexistent Hypoparathyroidism and Hyperthyroidism. Oliver Cope and Gordon A. Donaldson. 329.

Gastro-Intestinal Studies. (7) The Excretion of Xylose in Pernicious Anæmia. O. M. Helmer and Paul J. Fouts. 343.

The Significance of the Sodium and Potassium Content of Muscle Tissue and the Relation of the Amount of Edema Fluid in Muscle to the Level of Serum Protein in Experimental Nutritional Edema. W. B. McClure and Winifred Frans Hinman. 351.

The Composition of Human Gallbladder Bile and its Relationship to Cholelithiasis. John G. Reinhold, L. Kraeer Ferguson, and Ambrose Hunsberger. 367.

Concerning the Naturally Occurring Porphyrins. (5) Porphyrins of the Feces. Cecil James Watson. 383.

Pathology of Pregnancy Toxemias. Harry M. Zimmerman and John P. Peters. 397.

Renal Physiology in Lobar Pneumonia. Lee E. Farr and T. J. Abernethy. 421.

Studies of the Circulation in Pernicious Anemia. Harold J. Stewart, Norman F. Crane, and John E. Deitrick. 431.

Determination of Blood Ascorbic Acid. M. Pijoan and F. Klemperer. 433.

*Neutralization Tests in Poliomyelitis. Sera taken during the Acute and Convalescent Stages of the Disease and tested with a Passage Virus and a Strain isolated during the 1935 New York City Outbreak. Maurice Brodie, Alfred E. Fischer, and Maxwell Stillerman. 447.

Macrocytic Anemia in Pregnant Women with Vitamin B Deficiency. Katherine O'Shea Elsom with the technical assistance of Albert B. Sample. 463.

*Chronaximetric Examination in B Avitaminosis during Pregnancy. F. H. Lewy. 475.

Neutralization Tests in Poliomyelitis.—The sera of 82 paralytic, 32 non-paralytic, and three encephalitic cases was tested in the acute stage of the disease within one week of the onset. The sera of only 14 of the 82 paralytic cases neutralized the virus (strain F.1 monkey passage). Of the 32 non-paralytic cases tested in the acute stage the sera of 18 had neutralizing bodies. Of 39 paralytic cases, the sera taken during the convalescent period showed neutralizing bodies in only two. Again, only two cases of 24 who had no neutralizing bodies during the acute stage developed these in the succeeding 12 to 16 months. The authors state : " No evidence of a definite relationship was found between the presence of protective substances in serum and (1) resistance to poliomyelitis, (2) the diagnosis of the non-paralytic form of poliomyelitis, and (3) the degree of recovery from paralysis." (L. E. A.)

Chronaxie Readings during Pregnancy.—In a study of eight women throughout their pregnancy, the author has been able to determine that on a diet poor in vitamin B neurological signs might develop. These were chiefly in the nature of parasthesia. Measurements of chronaxie showed changes which along with the clinical signs improved on the giving of vitamin B. (L. E. A.)

JOURNAL OF COMPARATIVE NEUROLOGY

Vol. 66. No. 2. April 1937.

Bilateral Inequality in the Number of Sensory Neurons in the Trunk of Vertebrates. Enzo Delorenzi. 301.
*Innervation of the Intrinsic Muscles of the Eye of the Cat. Sam L. Clark. 307.
A Further Investigation of Auditory Cerebral Mechanisms. L. E. Wiley. 327.
Anatomical Relations of the Commissures of Meynert and Gudden in the Cat. Thomas A. Weaver, Jr. 333.
Diurnal Changes in the Retina of the Catfish, *Ameiurus nebulosus*. John H. Welsh and Clinton M. Osborn. 349.
Total Distribution of Taste Buds on the Tongue of the Kitten at Birth. Rush Elliott. 361.
The Telencephalon of *Tupinambis nigropunctatus*. I. Medial and Cortical Areas. Alice Osborne Curwen. 375.
Peripheral and Central Connections of the Upper Cervical Dorsal Root Ganglia in the Rhesus Monkey. Kendall B. Corbin, William H. Lhamon, and Donald W. Petit. 405.
The Function of the Brain in Auditory Localization. II. The Effect of Cortical Operation upon Original Learning. L. A. Pennington. 415.
A Cortical Lesion causing Cell Reaction in the Anteromedial Thalamic Nucleus. William H. Waller. 443.
Myelination in the Central Nervous System of the Albino Rat, treated with Thymus Extract. Albert C. Buckley. 449.
Cells and Fibres in Spinal Nerves. III. Is a 1 : 1 Ratio in the Dorsal Root the Rule ? J. F. Barnes and H. A. Davenport. 459.
The Mechanism of Vision. XIII. Cerebral Function in Discrimination of Brightness when Detail Vision is controlled. K. S. Lashley. 471.
Further Experimental Investigations on the Phenomenon of Homologous Response in Transplanted Amphibian Limbs. Paul Weiss.
II. Nerve Regeneration and the Innervation of Transplaned Limbs. 481.
III. Homologous Response in the Absence of Sensory Innervation. 537.

Innervation of the Intrinsic Eye Muscles.—The author, in a series of over 30 cats, performed one of the following operations on each animal : Removal of the superior cervical sympathetic ganglion and trunk, section of the ophthalmic and maxillary divisions of the trigeminal nerve, section of the oculomotor nerve, removal of the ciliary ganglion, and removal of the contents of the bulbous oculi. The animals were allowed to survive 5½ days to 3 weeks, and then the iris and ciliary body were sectioned and compared with those on the other side of the body. By the identification of degenerated fibres he has been able to show that the trigeminal nerve supplies no elements to these muscles, that the cervical sympathetic innervates only the dilator muscle of the iris, that fibres from the oculomotor nerve relayed in the ciliary ganglion supply the constrictor of the pupil and the ciliary muscle. Furthermore, each muscle fibre is supplied with a nerve-ending and no evidence was found of a terminal network or syncytium. Similar endings were found in the intramuscular connective tissue of the ciliary body, but the author does not believe that these subserve a proprioceptive function. (J. D.)

Vol. 67. No. 1. June 1937.

Structure of the Primary Olfactory Cortex of the Mouse. James L. O'Leary. 1.
The Function of the Brain in Auditory Localization. III. Post-operative Solution of an Auditory Problem. L. A. Pennington. 33.
Quantitative Studies of the Vagus Nerve in the Cat. II. The Ratio of the Jugular to Nodose Fibres. James O. Foley and Franklin S. Dubois. 69.
Structural Variations of the Visual Cortex in Primates. Gu Ngowyarg. 89.
*Function and Structure in the Chronically Isolated Lumbo-sacral Spinal Cord of the Dog. Sarah S. Tower. 109.
The Innervation of the Abdominal Chromaffin Tissue. W. Henry Hollinshed. 133.
Localization within the Cerebellum of Reactions to Faradic Cerebellar Stimulation. W. K. Hare, H. W. Magoun, and S. W. Ranson. 145.

Function and Structure in the Chronically Isolated Lumbo-sacral Spinal Cord.—The author isolated the lumbo-sacral cord in three 6-weeks puppies by sectioning the dorsal roots and transecting the cord above and below. Post-mortem examination was done at 2, 5, and 6 months. After operation the zone innervated by the isolated

cord showed abolition of all motor functions except those mediated by the sympathetic system. Later, quick jerky movements developed which were elicited by tapping over the spinal cord. They were thus the result of direct irritation of the isolated length of cord, but as they were co-ordinated it was demonstrated that an organizing mechanism existed at that level. This mechanism was, however, unable to stimulate itself, and the author concludes that autochthonous activity is not a property of the mammalian spinal cord. Histological examination showed evidence of continued growth of the cells and fibres. She states, therefore, that function is not necessary for the preservation of nerve cells and their processes. (J. D.)

JOURNAL OF MENTAL SCIENCE

Vol. 83. No. 344. May 1937.

An Inquiry into the Incidence of Neuropathic Conditions in the Relations of Normal Persons: Report by the Mental Deficiency Committee of the Royal Medico-Psychological Association. 247.
*Diffuse White Matter Gliosis in Mental Defectives. A. Meyer and N. C. Cook. 258.
Aspects of Temperament in Adolescent Male Offenders. H. T. P. Young. 268.
Endemic Bacillary Dysentery. J. J. B. Martin. 289.
*Hypnosis in Mental Hospital Practice. C. M. Copeland and E. Howard Kitching. 316.

Diffuse White Matter Gliosis.—As a result of examining 22 cases of mental deficiency, comprising seven showing gross neurological signs chiefly extrapyramidal, six microcephalics with spastic diplegia, two able-bodied microcephalics, one idiot, and six mongols, the authors draw attention to the proliferation of the fibrous glia. This was particularly noticeable in the cerebral and cerebellar white matter. It was often patchy, mainly perivascular, and occasionally in strip-like scars. The pathogenesis of the gliosis, however, remains obscure. Besides the gliosis, lesions of varying character were also found in a certain number of the subjects. (L. E. A.)

Hypnosis.—From a study of 40 cases to whom hypnosis was given, the authors form the opinion that the chief value of hypnosis is that it presents a more ready method of exploring the mental processes. They state that hypnosis alone was of no therapeutic value. (L. E. A.)

JOURNAL OF NERVOUS AND MENTAL DISEASE

Vol. 85. No. 5. May 1937.

The Dorsal Trigeminal Tract and the Centre Median Nucleus of Luys. James W. Papez and Wayne Rundles. 505.
The Institutional Management of Syphilis. Stanley R. Dean. 520.
The Effect of Stimulation of the Cortex Cerebri upon the Effector Mechanisms which mediate Movements of the Iris and Membrana Tympani. J. G. Bryne. 528.
Charcot-Marie-Tooth Disease with Primary Optic Atrophy. D. E. Schneider and M. M. Abeles. 541.
*Spontaneous Eidetic Imagery in a Case of Chronic Epidemic Encephalitis. H. B. Lang, Philip Polatin, and Sylvia Hotchkiss. 548.
Paramyoclonus Multiplex and Neurosyphilis. S. M. Weingrow. 557.

Spontaneous Eidetic Imagery in a Case of Chronic Epidemic Encephalitis.—A case is described in which attacks of spontaneous eidetic imagery occurred accompanied by intense anxiety and subjective unpleasant feelings in the eyes. No objective indications of oculogyric crises were observed. It is suggested that the eidetic imagery might be a consequence of the lesion due to the chronic encephalitis or it might be a release of a latent eidetic disposition or an alteration of the usual physiology of the sense perceptions, but no decision could be come to on this point.

Calcium alone and in conjunction with scopolamine, stramonium, and atropine was exhibited, and whether post or propter there followed a diminution of the anxiety, a fragmentation of the images, a lessening of detail, and a gradual fading. (R. G. G.)

Vol. 85. No. 6. June 1937.

Personality and Chronic Arthritis. Gotthard Cohen Booth. 637.
An Experimental Method for the Production of Decerebrate Rigidity in Dogs by Vascular Occlusion. Robert R. White. 663.
*The Reaction of Certain Psychotic Types to Alcohol. Carl E. Trapp and Purcell G. Schube. 668.
The Social Background of Occupational Neuroses. Ernest Harms. 689.
Sigmund Freud as a Neurologist. Smith Ely Jelliffe. 696.

Reaction of Certain Psychotic Types to Alcohol.—Doses of 40 per cent. alcohol up to a maximum of 180 c.c. were administered by a nasal tube to 21 psychotic cases. Of these 15 suffered from dementia præcox, one of which was simple, one hebephrenic, nine catatonic, and four paranoid; three were cases of psychosis with mental de-

ficiency ; and three, cases of manic depressive psychosis in the depressed phase.

Of the fifteen cases of dementia præcox the emotional state under alcohol of the simple type was unchanged ; of the hebephrenic patient his hebephrenic state was simply elaborated ; of the three cases of paranoid dementia præcox one became emotionally belligerent, whereas the others remained unchanged ; of the nine cases of catatonic dementia præcox five wept, displayed definite sorrow, but in three of the cases this was altered by episodes of silly laughter ; three of the nine cases remained indifferent ; and one exhibited erotic behaviour.

Of the mentally deficient cases all three exhibited marked depression and crying under alcohol.

Of the three manic depressive cases all three exhibited marked depression and crying under alcohol.

Negativism as exhibited by six of the dementia præcox cases did not change under alcohol.

With this dose no permanent therapeutic effect was noted, though temporary benefit was remarked in six. Mutism, which was marked in certain cases, was abolished for the time being and so the emotional content could be explored. It was possible to differentiate the catatonic stupors, who showed no emotional accompaniment of their narrative, from the depressive stupors, who showed a marked emotional reaction. (R. G. G.)

JOURNAL OF PHYSIOLOGY

Vol. 89. No. 3. April 1937.

The Electrical Responses of Light-adapted Frogs' Eyes to Monochromatic Stimuli. Ragnar Granit and C. M. Wrede. 239.

The Quantitative Estimation of Histamine in the Blood. C. F. Code. 257.

The Absorption of Amino Acids and their Distribution in the Body Fluids. Charles Bolton and G. Payling Wright. 269.

Vitamin B_1 and Fatty Livers. E. W. McHenry. 287.

The Chemical Agent in the Sympathetic Control of Retraction of the Nictitating Membrane of the Cat. J. Secker. 296.

The Insensitivity of the Cervix Uteri to Oxytocin. W. H. Newton. 309.

The Action of Certain Enzyme Poisons on the Frog's Auricle. 316.

Vol. 89. No. 4. June 1937.

The Absorption Spectra of Visual Purple and of Indicator Yellow. R. J. Lythgoe. 331.

Cholinesterase in Voluntary Frog's Muscle. A. Marnay and D. Nachmansohn. 359.

Cholinesterase in Invertebrate Muscles. Z. M. Bacq and D. Nachmansohn. 368.

*Observations in Man upon a Blood Pressure raising Reflex arising from the Voluntary Muscles. M. Alam and F. H. Smirk. 372.

Further Observations on the Action of Acetyl choline, Prostigmine, and Related Substances on the Knee-Jerk. A. Schweitzer and Samson Wright. 384.

Ætioporphyrin and Hæmoglobin Regeneration after Hæmorrhage. J. Howell Hughes and A. L. Latner. 403.

Afferent Fibres from the Stomach and Small Intestine. J. T. Irving, B. A. McSwiney, and S. F. Suffolk. 407.

A Simple Closed-circuit Metabolism Apparatus for Small Animals. W. H. Newton. 421.

The Cholinesterase in Sympathetic Ganglia. F. Th. v. Brücke. 429.

The Actions of Acetyl choline on Denervated Mammalian and Frog's Muscle. G. L. Brown. 438.

The Duration of the Recovery Period following Strenuous Exercise, measured to a Base Line of Steady, Mild Exercise. C. R. Cowan and O. M. Solandt. 462.

A Blood Pressure raising Reflex arising from the Voluntary Muscles.—By occluding the circulation to one limb and exercising that limb, it is possible to bring about a rise in blood pressure as measured in another limb. The rise in blood pressure continues after the cessation of exercise if the occlusion of circulation is not released. If on stopping the exercise the occlusion is released the blood pressure rapidly falls. These observations indicate that some substance accumulates in muscle during its activity and that this substance activates nerve endings in the muscle which send impulses centrally, thereby bringing about a rise in blood pressure. (L. E. A.)

L'ENCEPHALE

Vol. 32. Vol. 1. April 1937.

*Les Troubles Mentaux dans les Méningiomas de la Petite Aile du Sphénoide. (Mental symptoms in meningiomas of the small sphenoidal wing.) M. David and H. Askenasy. 169.

L'Etat Crépusculaire Hystérique. (Ganser.) (Twilight hysterical state.) W. Riese and A. Requet. 209.

Meningiomas of the Sphenoidal Wing.—The authors discuss mental symptoms associated with tumours in the region of the sphenoid. They find that confusion, impairment of memory, and disorientation are common symptoms. There is a change of character and a degree of puerilism ; these symptoms are very frequent, occurring in 84 per cent. of the cases, and are quite independent of the site of the tumour in over 50 per cent. of the cases. They found a degree of intellectual impairment, similar to that found with lesions of the frontal lobe. (D. D.-B.)

Vol. 32. Vol. 1. May 1937.

Recherches sur le Traitement des Maladies Mentales par L'Insuline. (Investigations on the treatment of mental illnesses by insulin.) G. Bychowski, M. Kaczynski, C. Konoplea and K. Szczytt. 233.
Les Remissions dans la Schizophrénie Statistique sur leur Fréquence et leur Durée avant L'Insulinethérapie. (Remissions of schizophrenia : statistics of their frequency and duration before insulin therapy.) L. Fromenty. 275.

Vol. 32. Vol. 2. June 1937.

*Les Polypeptides du Sang et du Liquide Céphalo-Rachidien dans la Paralysie Générale. (The polypeptides of the blood and cerebro-spinal fluid in general paralysis.) H. Claude, J. Dublineau, P. Masquin, and Mlle. Bonnard. 1.
Les Concepts Dynamiques et la Crise Epileptique (Dynamic concepts in epilepsy.) Smith Ely Jelliffe. 15.
*L'Epilepsie Réflexe. (Reflex epilepsy.) A. Redovici, M. Schachter, and S. Kisiler. 26.

Polypeptides of the Blood and Cerebrospinal Fluid.—The authors found that the polypeptides in the cerebrospinal fluid are often augmented in general paralysis of the insane. This appears in relationship to hepatic insufficiency or the correlation of the blood polypeptides. They have observed a fall of polypeptides in the cerebrospinal fluid as the case improves, and suggest that this test may be of prognostic value. (D. D.-B.)

Reflex Epilepsy.—The authors describe two cases of patients who have attacks of epilepsy closely associated with the taking of monobromide of camphor as a genito-urinary sedative. (D. D.-B.)

PHYSIOLOGICAL REVIEWS

Vol. 17. No. 2. April 1937.

The Male Sex Hormone. F. C. Koch. 153.
Rods, Cones, and the Chemical Basis of Vision. Selig Hecht. 239.
Formation and Destination of Red Blood Cells. Raphael Isaacs. 291.
The Nature of Uterine Contractility : A Survey of Different Trends. Samuel R. M. Reynolds. 304.

PSYCHIATRIC QUARTERLY

Vol. 11. No. 2. April 1937.

*Review of Activities of the Psychiatric Institute during the Past Year. Nolan D. C. Lewis. 181.
Pfropfschizophrenia. Pompeo Milici. 190.
Prolonged Sedation with Sodium Barbital. Philip Polatin. 213.
The Concept of Catatonia. George S. Sprague. 222.
Studies of Obsessive Ruminative Tension States. Lawrence F. Woolley. 237.
Dream Studies in Hallucinated Patients. Carl E. Trapp and Richard H. Lyons. 253.
Technical Approaches used in the Study and Treatment of Emotional Problems in Children. J. Louise Despert. 267.
Family Care of Patients at the Marcy State Hospital. Sidney W. Bisgrove. 296.
The Science of the Living Organism. Zygmunt Piotrowski. 305.

Review of Activities.—To research workers and those directing centres of investigation it is of advantage to know of work going on in large psychiatric institutions. Dr. Lewis outlines in this article the work being carried on in the various departments. It is evident from his report that the work covers a wide field. (I. C. H.)

REVUE NEUROLOGIQUE

Vol. 67. No. 4. April 1937.

*De L'Encéphalomyélite Nécrotique Subaiguë. (Subacute necrotic encephalomyelitis.) Riser, Geraud, and Planques. 455.
*Sur un Cas d'Hémichorée avec Lésion du Noyau Caudé. (A case of hemichorea with lesion of the caudate nucleus.) A. Austregesilo and A. Borges-Forte. 477.
*Parkinsonisme Traumatique. (Traumatic Parkinsonism.) Mathieu-Pierre Weil and V. Oumansky. 489.

Subacute Necrotic Encephalomyelitis—A patient of 29 years with progressive paraplegia for 2½ years associated with loss of all forms of sensation and later affection of the upper limbs. The autopsy showed a central necrosis of the spinal cord, syringomyelic cavity extending through the dorsal region, and dense arachnoiditis. Areas of perivascular hæmorrhage and œdema were found in the cerebral white matter, and in places a small amount of perivascular infiltration. On account of these cerebral changes, which are not very convincing, the authors consider the disease to be an encephalomyelitis rather than an unusually malignant type of syringomyelia. (D. D.-B.)

A Case of Hemichorea.—A patient of 70 years with progressive dementia of arteriosclerotic type had shown involuntary movements of the left upper limb and left side of the face for 2 years. The movements in the limb were in the nature of sudden spasms of inco-ordinated torsion. Autopsy showed a small zone of softening in the

middle third of the head of the right caudate nucleus. Lacunar degeneration of the remainder of the basal ganglia was present. No mention of the corpus sub-thalamicum of Luys is made, and in view of the frequent association of damage to this nucleus and hemichorea it is indeed doubtful if the movements in this case can be attributed to the caudate nucleus. (D. D.-B.)

Traumatic Parkinsonism.—Two cases of Parkinsonism are discussed in relation to trauma months earlier in one case and years earlier in the second. The patients are aged 29 and 28 years respectively. (D. D.-B.)

Vol. 67. No. 5. May 1937.

Amyotrophe Vraisemblablement Myopathique chez deux Jumelles Univitellines. (Similar myopathy in Uniovular twins.) Andre-Thomas, Paisseau, Sorrel, and Mme. Sorrel-Dejerine. 567.

Syndrome de l'Hémicone Médullaire par Hématomyélie. (One-sided conus lesion from hæmatomyelia.) E. Moniz and L. Pachego. 575.

*Myoclonies Palato-Pharyngo-Laryngées au Cours d'un Neurinome de la Région Latérobulbaire. (Palato-pharyngo-laryngeal myoclonus in the course of an eighth nerve tumour.) E. de Savitsch and R. A. Ley. 585.

Palato-pharyngo-laryngeal Myoclonus.—Autopsy revealed some hypertrophy of the olive usually associated with such myoclonus, and the authors conclude that the essential lesion was produced by damage to the dentate nuclei when the tumour had deeply invaded the cerebellum. The central segmental bundle, damage to which usually accounts for the myoclonus, was intact. (D. D.-B.)

Vol. 67. No. 6. June 1937.

*La Structure de la Moelle Epinière en Coupes Longitudinales. (The structure of the spinal cord in longitudinal sections.) L. Laruelle. 695.

*Gliome Périphérique de la Jambe d'Origine Neuro-vasculaire avec Crises de Sympathalgies Diffuses et Tenaces. (Peripheral neuro-vascular tumour in the leg, with symptoms of pain.) D. Paulian, I. Bistriceano, and C. Fortunesco. 726.

The Spinal Cord in Longitudinal Sections.—This is a preliminary report of a study of spinal cords in longitudinal sections. The cords examined were those of animals and man of various ages. The authors designate the cells in the grey matter according to their function—somatic or vegetative. In studying the columns of motor cells he finds that they may be grouped into pre- and post- fixed groups. He finds that the blood vessels run longitudinally in the cord and one vessel may supply a whole column of cells. This is especially so in the column of cells concerned with vegetative function. In this latter column he recognizes several centres, namely the ciliospinal, cardiac, splanchnic, and sacral parasympathetic centres. This work is of great interest and of considerable importance. (D. D.-B.)

Peripheral Neuro-Vascular Tumour in the Leg.—A small subcutaneous Schwannoma in the lower limb with a histological orientation to the walls of vessels. Touching the tumour caused widely radiating pains. This was a vascular type of Schwannoma. (D. D.-B.)

RIVISTA DI PATALOGIA NERVOSA E MENTALE

Vol. 49. No. 2. 1937.

Le Alterazioni del Sistema Nervosa Centrale Nella Lue Congenita Precoce con Particulare Riguardo al Comportamento dell'Ependima e dei Plessi Corioidei. (The alterations in the central nervous system in precocious congenital syphilis with particular reference to the behaviour of the ependyma of the choroid plexus.) A. Giordano. 213.

*Contributo alla Conoscenza del Problema Prognostico della Psicosi Ossessiva. (Contribution to the knowledge of prognosis in obsessive psychoses.) F. Cardona. 239.

Il Test di Rorschach nella Diagnostica Psichiatrica. (Rorschach's test in psychiatric diagnosis.) F. Cardona. 252.

Considerazioni sulla Prognosi Lontana nell' Amenza. (A consideration of the ultimate prognosis in amentia.) F. Cardona. 268.

*L'Atrofia Cerebrale Circoscritta o Malattia di Pick. (Circumscribed cerebral atrophy or Pick's disease.) R. Pasqualini. 273.

La Inversione Cromatica delle Cellule Basofile della Ipofisi e delle Cellule Principáli delle Paratiroidi. (The colour inversion in the basophil cells of the pituitary and of the principal cells of the parathyroid.) L. Severi. 333.

La Riattivazione del Liquor Nei Neuroluetici. (The reactions of the cerebrospinal fluid in cerebrospinal syphilis.) C. Rizzo. 241.

*Pneumoventriculogramma e Funzione Gastrica. Contributo Sperimentale allo Studio della Fisiopatologia dei Centri Encefalici Vegetativi. (Pneumo-ventriculograms and gastric function: An experimental contribution to the physiopathology of the vegetative centres in the brain.) L. Ricciteli and I. Lowenheim. 355.

Leptomeningite Essudativa Encefalica da Enterococco con Raccolte Saccate Simmetrische, Clinicamente Asintomatica. Morte Rapidissima. (Exudative cerebral leptomeningitis due to the enterococcus with localized symmetrical sacculation, clinically without symptoms, but with a very rapid fatal issue.) L. Bogliolo. 377.

Sulla Patogenesi della Forma Idrocefalica della Meningito Sierosa a Proposito di una Osservazione Anatomoclinica. (On the pathogeneses of the hydrocephalic form of serous meningitis illustrated by the anatomical and clinical observation of a case.) P. E. Maspes and U. Gallian. 401.

Prognosis in Obsessive Psychoses.—The author deprecates the exclusive study of the physico-chemical basis of the psychosis and pleads for a philosophical approach. He refers to the discussions in the literature of the association of obsessions with schizophrenia and describes a case of his own. He concludes that, although rare, the passage of an apparently simple obsessional case into a schizophrenia does occur. He thinks that prognosis should be based on an estimation of the schizoid characteristics of the individual patient, but even an absence of obvious signs of these does not exclude the possibility of a development of schizophrenia in an obsessional case. (R. G. G.)

Circumscribed Cerebral Atrophy.—The author, after having summarized the clinical and pathological picture as set out in the literature describes two cases of Pick's disease.

The first, studied only clinically, showed serious disturbances of volition and speech. These last were represented by an echolallia and in the advanced stages of the disease by a tendency to mutism. In addition, disturbances of mimicry were present, which finally resulted in complete inability to mimic. Also symptoms of lesions of the extrapyramidal system were observed. In view of these symptoms, a diagnosis was made of Pick's disease with fronto-striopallidal atrophy.

The second case had presented during life a massive dementia, absolute mutism, and, neurologically, moderate generalized hypertonia without signs of pyramidal lesions. Autopsy showed grave atrophy of the left temporal lobe and of the left supramarginal and angular gyrus. On macroscopical examination the frontal lobes, the rolandic region, the occipital lobes, the basal ganglion, and the right parietal and temporal regions appeared well conserved. The microscopical examination showed a diffuse atrophy of the gyri, minimal in the rolandic area and in the occipital lobes, and simple atrophy of medium degree in the remaining areas of the right hemisphere. In the left hemisphere the atrophy was definite throughout but maximal in the temporal lobe and in the supramarginal and angular gyri. The cortex in these last regions was seriously altered and especially so in the superficial strata, particularly in the third layer. The white matter was also altered. The neuroglial reaction was generally of moderate degree, perhaps on account of the slowness of the atrophic process, while the proliferation of the oligodendroglia was remarkable. Only in badly atrophied areas were there found cells with Alzheimer fibrillary changes. Silver-staining corpuscles and senile plaques were absent. No changes were found in the basal ganglia. (R. G. G.)

Ventriculograms and Gastric Function.—Experimental pneumo-ventriculograms in rabbits were found to cause changes in tone and gastric motility. After excluding all other explanations, the author assumes that these changes are due to disturbances of the vegetative centres in the brain. These results show the importance of the centres in the walls of the third ventricle and in the floor of the fourth ventricle in regulating the organs, and the authors consider that this proves that such regulation is indeed the function of these centres. (R. G. G.)

SCHWEIZER ARCHIV FÜR NEUROLOGIE UND PSYCHIATRIE

Vol. 38. No. 1. 1937.

*Beitrag zur Klinik und zur Histopathologie des Gangliozytoms der Medulla oblongata. (Contribution to the symptomatology and histopathology of the gangliocytomata of the medulla oblongata.) Erhard Amstad. 5.

Zur Pathogenese eines Falles von diffusem Gliom des Thalamus, des Aquäadukts und der Pinealgegend. (Contribution to the pathogenesis of a case of diffuse glioma of the thalamus, the aquæduct, and the pineal area.) Enrst Baasch. 26.

*Extension des Daumens : Ein neuer Pyramidenreflex. (Extension of the thumb : a new pyramidal reflex.) W. A. Bachtiarow. 45.

Die Stauungspapille. (Papillœdema.) Robert Bing. 49.

*Beiträge zur postembryonalen Entwicklung der Kleinhirnrinde beim Menschen. (Contributions to the postembryonic development of the cerebellar cortex in man.) M. L. Borowsky. 72.

Über die Methode einer doppelten Imprägnation des Nervengewebes. (A method of double impregnation of nervous tissue.) L. S. Goldin. 84.

*Familienkundliche, körperliche und psychopathologische Untersuchungen über eine Friedreich-Familie. (Genealogical, physical, and psychopathological investigations in a family with Friedreich's disease.) D. Klein. 89.

Zum Problem der Objektivierung und Projektion. (The problem of objectivation and projection.) Ch. de Monet. 117.

Schizophrenie bei Kretinismus. (Schizophrenia in cretinism.) von W. Plattner. 126.

Die klinische und biologische Bedeutung des Rossolimo'schen Reflexes. (The clinical and biological significance of Rossolimo's reflex.) J. F. Rittmeister. 142.

*Liquoruntersuchungen nach Commotio und Contusio cerebri. (Examination of the cerebrospinal fluid in cerebral commotion and confusion.) H. Schönstädt. 174.

*Die histologischen Veränderungen des Nervensystems bei experimenteller Avitaminose A, B (B_1) und G (B_2). (The histological changes of the nervous system in experimental avitaminosis A, (B_1), and G (B_2).) Z. M. Zimmerman. 195.

Gangliocytomata of Medulla Oblongata.—First a short account of the previous literature is given. The case reported here is that of a boy of 17 who had suffered from several attacks of pneumonia in early childhood. Since the first years at school he had had dyspnœa. hiccups, nausea, and

pain in the stomach, for which physical examination revealed no obvious cause. In the last half year of his life there was a severe exacerbation of symptoms, with difficulty in swallowing and loss of weight. He never complained of headache. There were no motor or sensory symptoms in the extremities. Pneumonia was the cause of death. At post mortem a tumour was found in the medulla oblongata extending from C1 to the lever of the striæ acusticæ. It consisted chiefly of fairly mature ganglion cells with Nissl bodies and nucleolus. Because of the absence of neurofibrils the term " gangliocytoma " is applied instead of ganglioneuroma. Different points in the histopathology are discussed. (K. S.)

New Pyramidal Reflex.—The examining person holds with one hand the patient's wrist ; with the other hand he strokes downward with a slight pressure the radial side of the forearm, starting from the lower end of the upper third. In normal persons or in patients with non-organic disease flexion of the end phalanx of the thumb results ; in patients with definite pyramidal signs in their lower extremities extension and abduction of the thumb results. In hemiplegias the symptom is only present in the early stage ; when contracture of the hand has developed the sign is absent. The author has seen it, however, in extrapyramidal diseases as well, and therefore he is not sure as to its diagnostic significance. (K. S.)

Cerebellar Cortex in Man.—The embryonic granular layer of the cerebellum disappears normally during the seventh to eighth month after birth. Even during early life some degenerated Purkinje cells may be seen in otherwise normal brains. These are supposed to be due to minor injuries or toxic conditions, for the Purkinje cells are vulnerable to injury which does not affect other elements. Many Purkinje cells may be seen migrating in the ventricular matrix and lamina ganglionaris within the first weeks of extra-uterine life. (K. S.)

Family with Friedreich's Disease.—In a family of six children three were normal and three showed the typical picture of Friedreich's disease combined with endocrine, mental disturbances, and deformities of the vertebral column. The disease was first noticed between the seventh and ninth year of life. The two girls showed disturbances of menstruation and the boy had obvious signs of hypogenitalism. The mental picture in two cases was chiefly that of intellectual debility. In one case (girl) there was a paranoid-hallucinatory syndrome with periods of great emotion and agitation. The genealogical aspect was most interesting. The descent of the parents, who were cousins, could be traced back to Frey's famous Friedreich family " Glaser " (1912), the complete clinical history of which is known back to the sixteenth century. The paper, which adds three cases to the 15 cases of the " Glaser " family, presents an important supplement to the best-studied family tree of Friedreich's disease in the literature. (K. S.)

Cerebrospinal Fluid in Trauma.—After a review of the previous literature on the subject, an account of 76 cases of cerebral commotion and contusion is given in which the cerebrospinal fluid had been examined. The examination was made 1 day to 10 years after the trauma. A complete examination of the cerebrospinal fluid was only done in 22 cases. The pressure was measured in all 76 cases. The pressure was increased in 66·7 per cent. of all cases examined between the 1st and 30th day after the trauma, and there is no marked difference between these cases and the cases examined later. Among the 22 fully examined cases 36·4 per cent. showed an increased number of cells. In 81·8 per cent. of these cases the Nonne-Appelt reaction was weakly positive ; both the Pandy and Weichbrodt reactions were positive in the majority of cases. The colloidal reactions were usually normal. The glucose was increased in only one case. About one-half of the cases showed normal chlorides ; in the others there was increase or decrease of chlorides. (K. S.)

Histology of Avitaminosis.—The conditions of the experiments have been improved, compared with previous ones, in so far as the food, apart from the absence of the vitamin, was identical with the natural food of the animals. In avitaminosis A in white rats the histological result was : myelin degeneration in the anterior and posterior roots of the spinal cord, in the brachial plexus and sciatic nerve, in the posterior columns and periphery of the cord. A sufficient amount of carotin, of which vitamin A is a derivative, prevents the changes if added to the food sufficiently early and in sufficient quantity. In avitaminosis B (B_1) in dogs the changes were : myelin degeneration in the sciatic, median, ulnar, vagi nerves and brachial plexus. They were least marked in the vagus nerve, most in the sciatic nerve. Previous experimental work on avitaminosis G (B_2) showed that the " black tongue " disease of dogs, which is similar to human pellagra, can be produced by a diet which lacks vitamin G (B_2). The diet had other deficiencies as well. With a perfectly correct diet which only lacked vitamin G (B_2) the author produced changes which were clinically and pathologically less similar to pellagra. This suggests that pellagra is not caused by a pure G (B_2) deficiency but by multiple deficiencies. The pathological changes observed in pure G (B_2) deficiency were : marked myelin degeneration of the peripheral nerves (including vagus nerve), myelin degeneration of the posterior, sometimes also of the

anterior roots and degeneration of the posterior columns with consecutive gliosis. The ganglion cells of the cortex and of the spinal cord were intact. Thus, the changes in the spinal cord and in the peripheral nerves observed in pellagra are identical with those produced by experimental G (B_2) deficiency. All the other changes of the central nervous system and the body observed in this disease seem to be due to other deficiencies of the food, the nature of which is not known. (K. S.)

ZEITSCHRIFT FÜR DIE GESAMTE NEUROLOGIE UND PSYCHIATRIE

Vol. 58. 1937.

Contains a report on the second annual meeting of the Society of German Neurologists and Psychiatrists at Frankfurt from 22nd to 25th August, 1936. Two main topics were discussed, namely: blood disease and central nervous system; and the significance of the pathology of the brain for neurology and psychiatry. Other more or less related subjects were also dealt with. Many papers, particularly the opening one, are unsuitable for abstracting, because they are either critical reviews of more general nature or are abstracts themselves. Attention may be drawn, however, to the following papers:

Die Pathologie des Nervensystems bei Blutkrankheiten. (Pathology of the nervous system in blood diseases.) G. Bodechtel. 48.

Die Bedeutung der Porphrine für die Pathogenese gewisser neurologischer Krankheitsbilder. (The significance of the porphyrin compounds in certain nervous diseases.) A. Bingel. 79.

*Weitere Untersuchungen zum Wilson-Pseudosclerose Problem. (Further investigations on the problem of Wilson's disease.) 92.

Gefass und Nervenzelle. (Blood vessels and nerve cells.) E. Scharrer. 93.

Optische Untersuchungsmethoden und Liquordiagnostik. (Optical methods in examining the cerebrospinal fluid.) K. F. Scheid. 101.

*Liquoruntersuchungen bei destructiven Erkrankungen des Nervensystems, besonders bei Schizophrenie. (Investigation of the cerebrospinal fluid in destructive diseases of the central nervous system, especially schizophrenia.) H. Lehmann-Facius. 109.

Zur Pathophysiologie psychogener Bewegungsstörungen. (The pathophysiology of movement in psychogenic disturbances.) H. Altenburger. 133.

Bericht über die Gehirnpathologie in ihrer Bedeutung für Neurologie und Psychiatrie. (A survey of brain pathology. Its significance to neurology and psychiatry.) K. Kleist. 159.

Sensorische Aphasien. (Sensory aphasias.) E. Beck. 193.

*Über die Bedeutung der basalen Rinds. Auf Grund von Beobachtungen bei Pickscher Krankheit und bei gedeckten Hirnverletzungen. (The significance of the basal cortex, based upon observations on Pick's disease and head injuries.) H. Spatz. 208.

Über pathomorphologische und methodologische Voraussetzungen für die Hirnlokalisation. (Conditions requisite for localization of lesions in the brain.) W. Scholz. 234.

Gibt es eine pathologische Anatomie der Schizophrenie? (Is there a morbid anatomy of schizophrenia?) Gerel Peters. 239.

The Problem of Wilson's Disease.—The author describes in the livers of patients affected by hepatico-lenticular degeneration changes of the nuclei which he considers to be comparable to the nuclear changes commonly found in the so-called Alzheimer's glia cells. Glycogen is found histochemically which is missing in the glia cells, possibly owing to rapid breakdown.

The Cerebrospinal Fluid in Destructive Diseases of the Central Nervous System.—If a mixture of brain phosphatides plus Meinicke extract is added to cerebrospinal fluid, a precipitation is seen under certain circumstances; 310 cerebrospinal fluids have been investigated by this method. Of 56 cases of dementia præcox 49 showed a positive result; of 153 organic conditions 14 and of 101 psychoneuroses and functional psychoses only five were positive.

Significance of the Basal Cortex.—Change of personality is related to pathological lesions in the orbital part of the frontal and temporal lobes in various conditions (meningioma, Pick's disease, head injury, in the latter particularly in cases of contrecoup). Details must be read in the original paper. (A. M.)

Vol. 59. No. 1. June 1937.

Eine diagnostische Untersuchung Rorschachs auf Grund der Helldunkeldeutungen ergänzt. (An addition to Rorschach's test.) Ernst Schneider. 1.

Die Erblage in der nächsten Verwandtschaft von 30 Fällen klimakterischer bzw. involutiver Melancholie. (A genealogical study of near relations of 30 cases of involutional melancholia.) Helene Schnitzenberger. 11.

Chronaxie und Zuckungsablauf beim menschlichen Muskel. (Chronaxie and muscle movement in man.) Heinz Schiffer. 24.

*Zur Frage der "Koagulationsnekrose" im Zentralnervensystem. (On the problem of coagulation necrosis in the central nervous system.) T. Markiewicz. 27.

*Zur Frage der "kolloiden" Degeneration und Ähnlicher Vorgänge im Zentralnerven system. (On the problem of colloid degeneration and related phenomena in the central nervous system.) T. Markiewicz. 53.

Über anthropometrische Indices zur Unterscheidung von Körperbautypen. (The anthropometric indices in somatic constitutional types.) Erik Strömgren. 75.

Zahl und Zählung der zelligen Elemente in der Spinalflüssigkeit. (The cell-count in the cerebrospinal fluid.) H. Jessen. 82.

Über die humoralen Veränderungen bei der Behandlung der Schizophrenie mit der Dauernarkose (Cloettan). (Hormonal changes during the

prolonged narcosis treatment of schizophrenia.) A. E. Kulkow, W. J. Weiland, and B. E. Kakusina. 96.

Zur Belastungsstatistik der Durchschnittsbevölkerung. Untersuchungen an den Familien eines niederschlesischen Krankenhausmaterials. (A statistical medical survey of the average population as obtained in lower Silesia.) Helmut Bormann. 110.

*Über eine adulte, sich bis ins Präsenium hinziehende Form der familiären amaurotischen Idiotie. (A case of amaurotic idiocy in an adult lasting until the presenium.) L. van Bogaert and Pierre Borremans. 136.

Über ein stummes, parasagittales Meningeom des Scheitellappens. (Asymptomatic parietal parasagittal meningeoma.) C. M. Behrend and E. Schilf. 158.

Coagulation Necrosis. Colloid Degeneration.—The two papers are reviewed together because in both an attempt is made to consider the degeneration in the central nervous system in the light of the laws of colloidal chemistry. The coagulation necrosis is strictly separated from common types of softenings on the strength of its appearance and the environmental reaction. The colloid degeneration is compared with amyloid degeneration. The significance of the vascular factor is stressed. Colloid degeneration and coagulation necrosis are essentially different. Details of the thorough investigations must be read in the original paper. (A. M.)

Case of Amaurotic Idiocy.—Detailed clinical and histological report on a condition, beginning at the age of 15 with epileptic fits and with gradual manifestation of extrapyramidal disturbances, paresis, and mental deterioration; death at the age of 62 due to marasmus. Histologically typical ballooning of the ganglion cells, which contain lipoidal inclusions. The case greatly resembles even in detail earlier observations, particularly those of Kufs and Meyer. As in these latter cases, visual disturbances were completely lacking. The general importance of this late form of amaurotic idiocy is emphasized. (A. M.)

ZENTRALBLATT FÜR NEUROCHIRURGIE

Vol. 2. No. 3. April 1937.

Das Ependymom der Grosshirnhemisphären im Jugendalter. (Ependymoma of the cerebrahemisphere in youth.) W. Tünnis and K. J. Zülch. 141.

*Carotid-Cavernous Aneurysms. (Pulsating Exophthalmos.) W. E. Dandy. 165.

Zur Genese der Monroi-Cysten. (On the genesis of "Monroi" cysts.) F. K. Kessel. 206.

Carotid-cavernous Aneurysms.—This is the concluding article of two. The author has dealt with all aspects of the condition. It requires detailed study. (H. C. I.)

Vol. 2. No. 4. June 1937.

*Déformations des sinus droit et longitudinal inferior et des veines profondes du cerveau dans le diagnostic des néoplasmes cérébrales. (The significance of deformities of the right and inferior longitudinal sinuses and of the deep cerebral veins of the brain in cerebral tumour.) E. Moniz. 214.

*Über die diagnostische Bedeutung röntgenoligisch darstellbarer Kalkherde in den Grosshirnhemisphären von Erwachsenen nebst Bemerkungen zur Klinik und Pathologie des Oligodendroglioms. (The diagnostic significance of calcified areas in the cerebral hemispheres of adults as shown by X-ray: observations on the symptomatology and pathology of oligodendrogliomata.) St. Kornyey. 224.

Spontaneous Intracerebellar Hæmorrhage treated by Operation. A. Torkildsen. 242.

Deformities of the Right and Inferior Longitudinal Sinuses.—After the X-ray photograph is taken to demonstrate the filling of arteries by thorotrast, further films are taken. In these it may be possible to demonstrate abnormalities of the venous channels. This may be of especial value in space-occupying lesions lying posteriorly or in the posterior fossa. (H. C. I.)

Calcified Areas in the Cerebral Hemispheres.—In 107 histologically confirmed gliomas of the cerebral hemispheres in adults, nine showed calcification by X-rays. All tumours with calcification turned out to be oligodendrogliomata. No tumour of this character occurred without calcification. (H. C. I.)

BOOK REVIEWS

PATHOLOGY OF THE CENTRAL NERVOUS SYSTEM

A study based upon a survey of lesions found in a series of fifteen thousand autopsies

Cyril B. Courville

(Pacific Press, California. 1937. 26*s*.)

As the title indicates, the book is based upon the large experience obtained in about fifteen thousand autopsies which have been evaluated by the author and his pupils. It may be for this reason that expectations are extremely high. If allowance is made for the objective of the book in attempting to present the gross " Kinetic " aspects of brain pathology rather than histological detail, the impression after reading the book is not wholly satisfactory. It may be conceded that some of the chapters on gross pathology, particularly those dealing with tumours, diseases of the intracranial blood vessels, infectious and traumatic diseases, are good and give much useful and in places original information. It is doubtful, however, whether the neglect of histological detail can be carried so far as to present vague statements. For it is stated that in general paralysis the iron is found in ganglion cells ; no mention is made of the cellular elements in which it is really deposited. It is also stated that the well-known rod cells are derived from the interfasicular glia. Again, Pick's disease is characterized as a disease of the white matter. It is suggested that the characteristic feature of " Pelizæus-Merzbacher's disease " is a perivascular demyelinization. It is hoped that in a future edition a close revision of the histological matter will be undertaken.

PRÉCIS D'ANATOMO-PHYSIOLOGIE NORMALE ET PATHOLOGIQUE DU SYSTÈME NERVEUX CENTRAL

Pierre Pasquin and J. O. Trelles

(Gaston Doin et Cie., Paris. 1937. 165 frs.)

The authors have succeeded in producing a readable summary of all the detailed anatomy of the nervous system. Each chapter has a brief section devoted to a summary of the experimental physiology of the region concerned and another to description of syndromes. Every conceivable nervous pathway is described and the excellent diagrams have the traditional clarity of French text-books. The views expressed are modern and a well-selected list of bibliography is given for each section. The book can be recommended as a reference book, though it is too full for the ordinary student, and its brief and schematic description does not allow much discrimination between what is useful and well-founded and what is obscure and semi-hypothetical. As an attempt to present the anatomy, physiology, and pathology of the nervous system all under one cover, it presents too ambitious a survey of anatomy to be found ideal by clinical neurologists.

PRACTICAL NEURO-ANATOMY

G. H. Globus

(Baillière, Tindall and Cox, London. 1937. 27*s*.)

This is essentially a book to be used in the practical class-room where facilities for dissection and for the study of microscope slides are available. The arrangement of the matter is such that the student is allotted a series of dissections to undertake, and, provided the student adhers to the directions, he will be amply rewarded by a clear and lucid description of the structures of the nervous system. The author, however, has gone further and included some chapters on the physiology of the nervous system. While welcoming any attempt to relate function and structure, it is doubtful if the author has been able to do himself full justice within the limits of the present book. The desirability of including in a book on practical neuro-anatomy nine pages devoted to a description of clinical examples of syndromes is open to question. The author is to be complimented on the style of printing and clear drawings. The photographs of sections through the brain stem and medulla are smaller than those of the spinal cord. This is unfortunate, as the structure of the former is more complicated than the latter and requires more detailed study.

THE DIAGNOSIS OF NERVOUS DISEASE

Sir James Purves-Stewart

(8th Edition. Edward Arnold & Co., London. 1937. 35*s*.)

This book already belongs to the group of standard works in clinical neurology which are indispensable both for students and specialists. With its comprehensive anatomical and physiological outlook and the clinical and theoretical details, it represents much more than a mere text-book of the symptomatology of nervous diseases. In this edition the knowledge gained in neurology in the last six years has been added.

PERSPECTIVES IN BIOCHEMISTRY

F. G. Hopkins

(Cambridge University Press, London. 1937. 15*s*.)

Collected in this volume are 31 essays prepared by past and present members of Sir Gowland Hopkins' laboratory. Each essay is in itself a wealth of knowledge expressing the view-point of the author in a given subject. Happily, the subjects chosen have allowed a play of expression by the individual authors and cover a wide range. Philosophical thought is encountered in all and as a source of stimulation for future workers this volume is indeed rich. Clearly printed, delightfully bound, this is a volume worthy of its purpose—a presentation to Sir Gowland Hopkins. It is to be hoped that it will form a standard for presentation volumes; it could not be bettered.

SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM

G. E. Gask and J. Paterson Ross

(Baillière, Tindall and Cox, London. 1937. 16*s*.)

This is a second edition of a useful book. The authors continue to express in guarded terms the indications for surgical intervention in a variety of maladies. The student with an enquiring mind will find much to stimulate him. As a balanced statement of the present position of the surgery of the sympathetic system, the book is to be commended.

TUMOURS OF THE CENTRAL NERVOUS SYSTEM

An investigation of the most recent advances. Association for Research in Nervous and Mental Disease. Proceedings of the Association, New York, 27th and 28th December, 1935.

(The Williams Wilkins Co., Baltimore. 1937. 34*s*.)

The book contains 16 papers which cover a wide aspect of the subject. Most papers not only refer to original work done by the authors but also give a general survey on the special theme. In this way the book represents an excellent review on the tumour problem. It is not possible in the course of a review to give this volume adequate consideration. As a source of valuable information the book stands out pre-eminent.

MEDICO-LEGAL ASPECTS OF THE RUXTON CASE

John Glaister. James Couper Brash

(E. and S. Livingstone, Edinburgh. 1937. 21*s*.)

Although this work deals solely with the medico-legal investigations in one notorious murder case, it should be read by everyone concerned in any way or even interested in medico-legal work. The book presents more clearly than ever has been done before the close co-operation necessary between the many medical and scientific experts and the police, in order that such a case may be brought to a successful conclusion. The probable identification of the dismembered remains by an anatomist assisted by a photographer, the work of an entomologist in determining the probable time of exposure of the remains by recognition of the larvæ present are unique in medico-legal literature.

In the less unusual sections, such as the identification of blood-stains, the book gives an example of thoroughness which should be studied carefully by anyone likely to be concerned in any important medico-legal investigation.

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